ASSESSMENT OF CARDIAC FUNCTION IN PATIENTS OF CHRONIC RENAL FAILURE BY ECHOCARDIOGRAPHY

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CHENNAI

CERTIFICATE

This is to certify that this dissertation entitled ***"ASSESSMENT OF CARDIAC FUNCTION IN PATIENTS OF CHRONIC RENAL FAILURE BY ECHO CARDIOGRAPHY"*** *submitted by* ***Dr. V. PAVANASAKUMAR*** *to the Faculty of General Medicine, The Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the requirement for the award of M.D. Degree Branch I (General Medicine) is a bonafide research work carried out by him under our direct supervision and guidance.*

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DECLARATION

I, ***Dr. V. Pavanasakumar****, solemnly declare that the dissertation titled* **"ASSESSMENT OF CARDIAC FUNCTION IN PATIENTS OF CHRONIC RENAL FAILURE BY ECHO CARDIOGRAPHY"** *has been prepared by me.*

This is submitted to the Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the M.D. Degree Branch I (General Medicine).

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INTRODUCTION

INTRODUCTION

Chronic renal failure (CRF) is associated with increased morbidity and mortality. Chronic renal failure affects almost all systems of the body and results in various abnormalities. Among the various causes, infections and cardiovascular causes contribute towards the large proportion of increased morbidity and mortality.

Cardiac disease is the major cause of death in CRF patients accounting for 40% of deaths in international registries.

In 1997, annual report of US Renal Data System (USRDS) revealed that morbidity in patients with CRF is attributed mainly to cardiac causes which account for 49% of the cases.

In the cardiovascular system, left ventricular hypertrophy (LVH) is the most frequent finding. The prevalence of left ventricular systolic and diastolic dysfunction is less clear.

Cardiac disease frequently predates the start of dialysis and LVH is common in moderate to severe chronic renal failure.

Echocardiography is one of the non invasive and sensitive tests for evaluating various cardiac changes in CRF patients at an early

stage. Echocardiography should be performed early in the course of CRF and may be valuable in the monitoring of therapy of these patients. Cardiac dysfunction is the major impediment to rehabilitation.

Patients in developing countries are managed mainly on conservative therapy and therefore, suffer from chronic acidosis, malnutrition, anaemia and azotemia. These factors further aggravate the cardiac dysfunction in uraemic patients.

The severe cardiac dysfunction is frequently noted in individuals around the time of commencement of dialysis, but there is little information on the prevalence and natural history of cardiac function in patients with milder degrees of chronic renal failure.

The present study was aimed at assessing the prevalence of systolic and diastolic dysfunction by echocardiography in patients with varying degrees of chronic renal failure who had been on conservative management.

Eighty percent to 90% of patients with chronic kidney disease experience hypertension during the course of the disease. Uncontrolled hypertension accelerates the rate of progression regardless of the cause of renal failure. Hence this study has also been used to compare the various cardiac changes in hypertensive CRF patients with normotensive CRF patients.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Cardio vascular aspects of chronic renal failure (CRF)

Background

Chronic Kidney disease is a pathophysiologic process with multiple etiologies, resulting in the inexorable attrition of nephron number and function and frequently leading to end stage renal disease (ESRD).

The definition of CRF requires that the pathophysiologic process described above last more than 3 months. A recently widely accepted international classification divides CRF into a number of stages defined by clinical estimation of the Glomerular filtration rate (GFR).

Table : Stages of chronic kidney disease

Stage	Description	eGFR, ml/min per 1.73 m^2
	* At increased risk	**90 (with CRF risk factors)**
1.	Kidney damage with normal or increased GFR	90
2.	Kidney damage with mildly decreased GFR	60-89
3.	Moderately decreased GFR	30-59
4.	Severely decreased GFR	15-29
5.	Renal failure/ESRD	< 15 (or dialysis)

For purposes of staging CRF, Current guidelines recommend estimating GFR using one of the two equations shown as below.

Recommended equation for Estimation of Glomerular Filtration Rate from Laboratory – Validated Plasma Creatinine Concentration (P_{cr})

Equation from the Modification of Diet in Renal Disease Study (MDRD)

Estimated GFR (ml/min per 1.73 m^2)

= $1.86 \times (P_{cr})^{-1.154} \times (age)^{-0.203}$

Multiply by 0.742 for women

Multiply by 1.21 for blacks

Cockcroft – Gault equation

Estimated Creatinine clearance (ml/min)

$$= \frac{(140\text{-age}) \times \text{body weight (kg)}}{72 \times P_{cr}\ (\text{mg/dl})}$$

Multiply by 0.85 for women

A common definition for CRF stipulates an eGFR of less than 60 ml/min/1.73m^2 or the presence of albuminuria, defined as an albumin to creatinine ratio greater than 30 mg/gm on a spot urine sample. Although with normative aging (age 20 to 80), the eGFR declines from

about 130 to 60 ml/min/1.73 m^2, a variety of pathobiological processes appear to begin when the eGFR drops below 60 ml/min/1.73m^2.

Most of the cardiovascular derangements occur below an eGFR of 60ml/min/1.73m^2, which roughly corresponds to a serum creatinine (Cr) greater than 1.5 mg/dl in the general population.

Approximate prevalence (%) of cardiovascular disease in the general population and in patients with chronic kidney disease

Population	Coronary Artery Disease (Symptomatic)	Left ventricular hypertrophy (Echocardiogram)	Cardiac failure (Symptomatic)
General population	5-12	20	5
Predialysis	NA	25-50	NA
Hemodialysis	42	75	40
Peritoneal analysis	40	75	40
Renal transplantation	15	50	10

*NA not available

Most clinical consequences of cardiac disease result from cardiomyopathy (or) ischemic heart disease. Cardiomyopathy may manifest as an enlarged, dilated left ventricle (LV) with systolic

dysfunction (or) as a hypertrophic ventricle with diastolic dysfunction, with or without myocardial ischemia.

It has also become evident that the structure of large arteries can be altered, not only by atherogenesis, but also by arteriosclerosis. Intramural vascular remodeling occurs as a consequence of sustained hemodynamic overload, with an increase in vessel stiffness and diameter.

There is a persistent and complex interplay of destructive vascular events and myocyte dysfunction that, if unrecognized, ultimately results in cardiac failure and death.

Arteriosclerosis contributes directly to ischemic symptoms, LV hypertrophy and systolic dysfunction by increasing cardiac workload. LV hypertrophy occurs in most of the severe renal failure cases and there is diastolic dysfunction with or without normal systolic function.

Cardiac disease can also result from the development of valvular heart disease. Most valvular lesions observed in patients with CRF are acquired and develop from dystrophic calcifications of the valvular annulus and leaflets, particularly the aortic and mitral valves. Such calcification is now known to be present far more frequently than previously recognized, with a prevalence of upto 55%, for the aortic

valves and 39% for the mitral valves (Foley RN, Parfrey PS et al, Am J Kidney Dis, 1998 and London GM et al J Am Soc Nephrol, 2000).

LV Hypertrophy is already evident in 40% of patients with moderate renal insufficiency and in 75% of those commencing dialysis (Levin A, Singer J, Thompson CR, et al, 1995). Both forms of LV hypertrophy (concentric and eccentric) are associated with an increased mortality risk in CRF patients.

Associated risk factors, including hypertension, diabetes, tobacco – use and anemia predispose to the much more rapid development of symptomatic cardiomyopathy. (Levin A, Thompson CR, 1999).

Such patients display a higher incidence of cardiovascular abnormalities at an earlier stage of CRF and at a younger age, often becoming symptomatic or exhibiting significant morbidity well before reaching end stage kidney function.

PATHOLOGY AND PATHOPHYSIOLOGY

LV Hypertrophy is an adaptive process that occurs in response to a long term increase in myocardial work caused by LV pressure or

volume overload; it results from the interactions among mechanical stimuli, locally generated growth factors and vasoactive substances.

LV tensile wall stress (σ), according to the law of Laplace, relates directly to the intraventricular pressure (P) generated and to the internal radius (r) of the ventricular cavity. It is inversely proportional to the ventricular wall thickness (θ).

$$\sigma = Pr / 2\theta$$

Therefore, the wall tension at any given pressure increases with the radius and vice versa. Conversely, as pressure (or) cavity volume (or both) is increased, there is an increase in wall thickness which reduces the systolic tension (and hence oxygen consumption) that must be developed by each myocyte. It is this wall remodeling that results in LV hypertrophy. The initial effects of LV hypertrophy are beneficial.

Eventually, however LV hypertrophy becomes maladaptive, with a sustained imbalance between energy expenditure and production, resulting in a chronic energy deficit and myocyte death. Within the myocardium, overstretching of papillary muscles is coupled with oxidant stress, apoptosis, architectural rearrangement of myocytes, and impairment in force development of myocardium (Cheng W, Li B,

Kajstura) et al; Stretch induced programmed myocyte cell death, 1995).

There is also evidence that abnormal expression of proto-oncogenes promotes the development of, particularly, fibroblasts, with an increase in extracellular collagen matrix and hence the development of myocardial fibrosis.

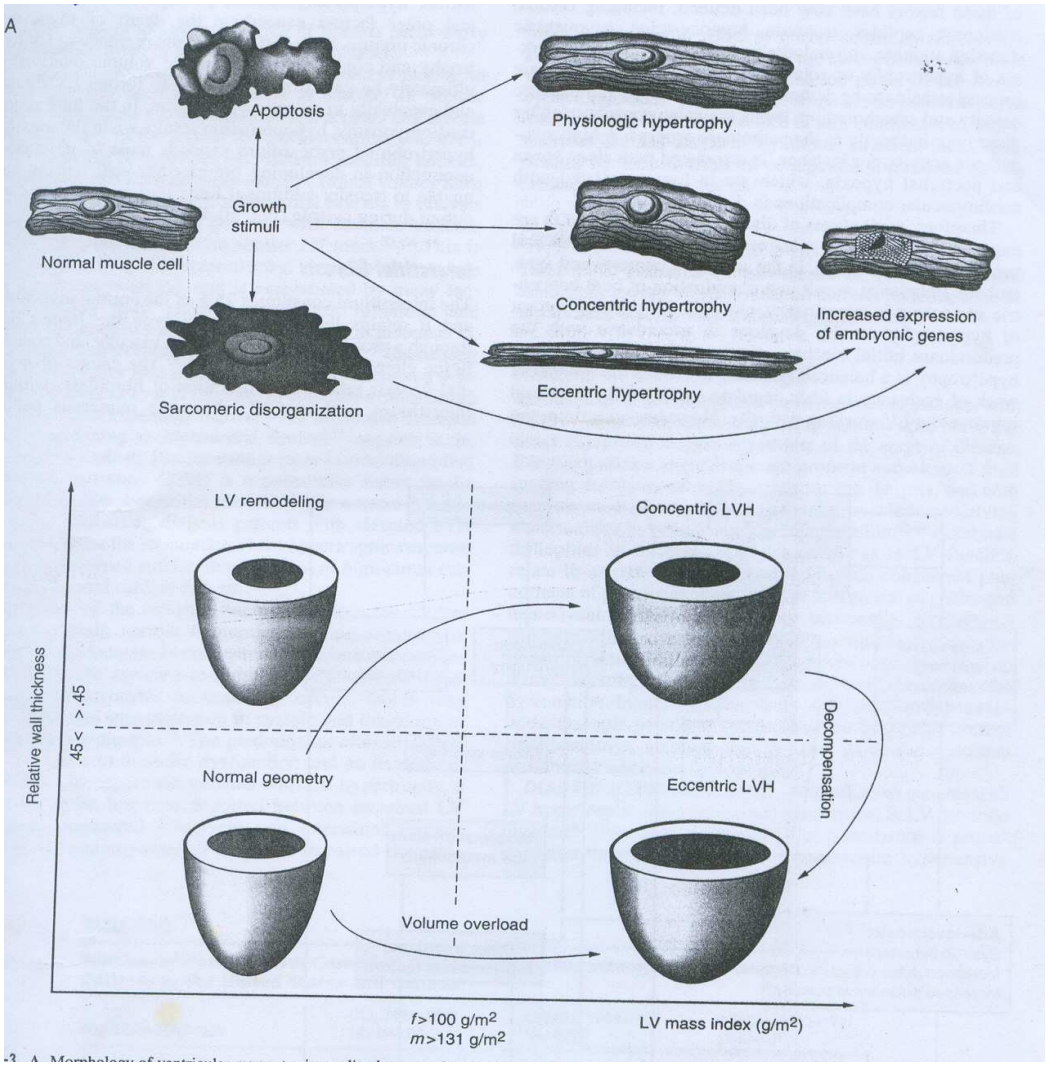
The consequences of these alterations are electrophysiologic abnormalities and maintenance of systolic efficiency at the expense of impaired diastolic filling. Arrhythmias are caused partly by conduction aberrations secondary to fibrosis and partly by prolongation of the action potential due to a slower reuptake of calcium by the sarcoplasmic reticulum. The latter, together with fibrotic change and increased LV wall stiffness, contributes substantially to abnormal diastolic function.

As a result, the geometry of the heart changes from concentric LV hypertrophy with normal LV volumes to eccentric LV hypertrophy with dilatation, the end stage of this tendency being severe LV dilatation with systolic dysfunction.

Eventually, in conditions of chronic and sustained overload, the deleterious effects of hypertrophy, increased LV chamber pressure,

and fibrosis dominate, leading to the development of cardiomyopathy and LV failure (Katz AM : Ann Intern Med, 1994).

Morphology of ventricular myocytes in cardiac hypertrophy and failure



Functional Abnormalities

Assessment of LV functional abnormalities in patients with CRF is often difficult.

Absence of symptoms does not imply intact functional reserve regardless of the stage of disease. The difficulties can be encountered when trying to distinguish clinically between systolic and diastolic dysfunction since multiple pathologic conditions are frequently present simultaneously, although usually one particular condition predominates in a clinical context.

Diastolic Dysfunction

The degree of disturbance is probably more than that observed in patients with hypertensive heart disease but milder than in those with hypertrophic cardiomyopathy. The abnormal ventricular filling in uremia results from increased LV stiffness caused by intramyocardial fibrosis and associated delayed relaxation. By virtue of an increase in LV stiffness, small changes in volume result in large changes in LV pressure, predisposing to symptomatic pulmonary edema. The reverse is also true : volume depletion results in a large fall in LV pressure with symptomatic hypotension and hemodynamic instability. (Ritz E, Rambausek M, et al, 1990) This is often the presenting feature of diastolic dysfunction.

Systolic Dysfunction

Resting systolic function is usually normal or even increased in patients with advanced renal disease in the absence of antecedent cardiac disease. However, decreased systolic function is frequently observed in patients in whom cardiac disease was present before the onset of dialysis therapy and in patients who have experienced prolonged and marked hemodynamic overload. Approximately 15% of patients have systolic dysfunction by the time they start dialysis. (Foley RN, Parfrey PS et al, 1995) Diminished myocardial contractility may also be a result of overload cardiomyopathy, in which the myocardium relies on starling forces to maintain a normal output. The manifestations of cardiomyopathy has a substantially worse prognosis than that for either concentric LV hypertrophy or LV dilatation with normal systolic function.

In dialysis patients, systolic dysfunction is strongly associated with the presence of ischemic heart disease or sustained biomechanical stress or both. However, it can also be a reversible manifestation of severe uraemia, abating when the uremic environment is removed. Uremic serum has been found to reduce the force of contraction of cultured myocytes in a concentration – dependent manner. (Weusenee D Low – Friedrich I et al : Invitro approach to “Uremic Cardiomyopathy”, Nephron, - 1993).

Renal transplantation has also been shown to normalize systolic function in dialysis patients with systolic dysfunction and subsequently to reduce but not normalize LV mass index.

Symptomatic Heart Failure

LV failure is a clinical condition that can be defined as the inability of the heart to maintain sufficient output to meet metabolic demands at rest, or the ability to maintain such demands only at the expense of a sufficiently raised venous pressure to result in pulmonary edema. If left untreated, myocardial hypertrophy can be viewed as an early milestone in the clinical course of cardiac failure.

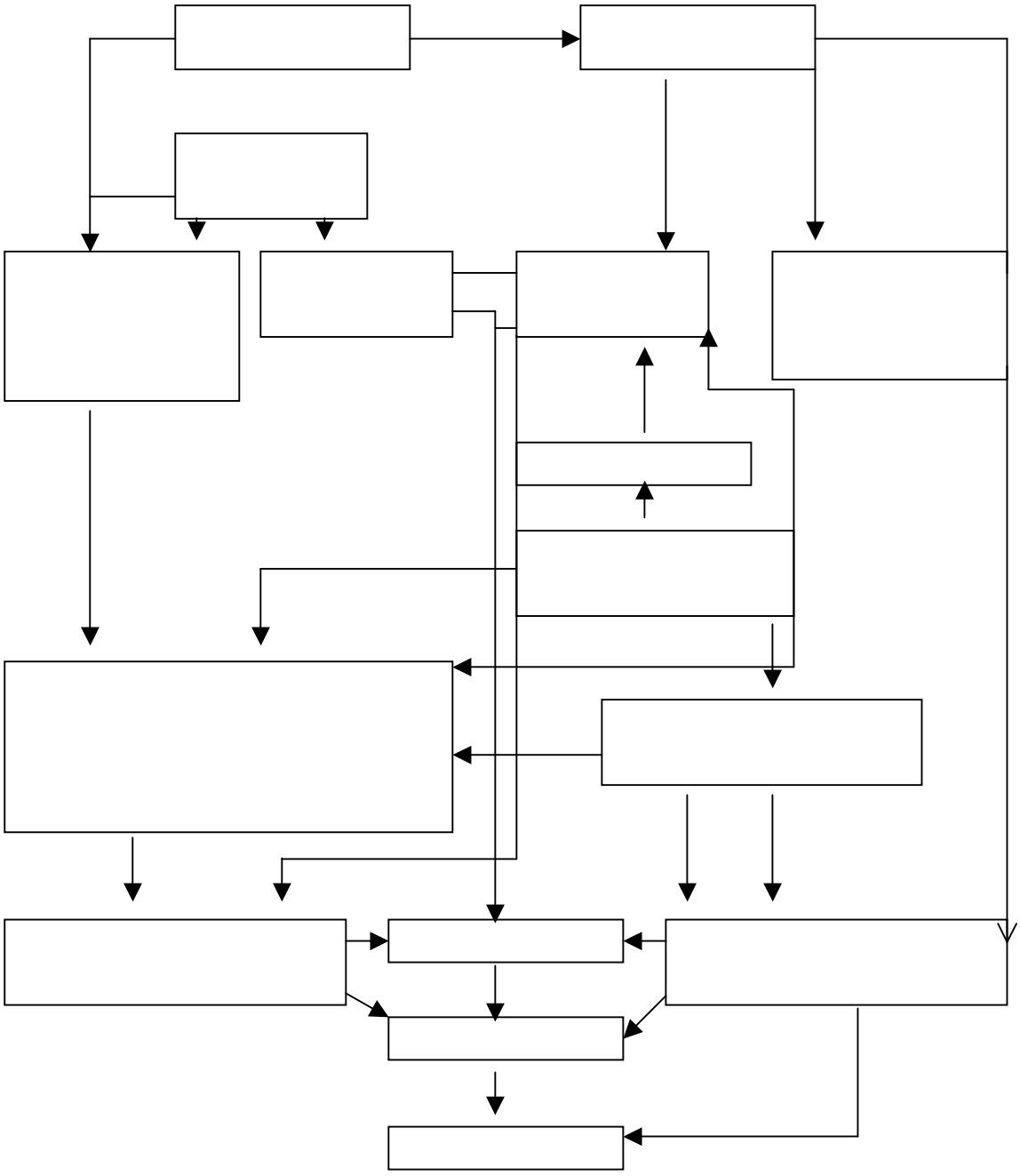
In a Canadian Cohort study, 275 patients had serial echocardiograms on starting dialysis and after 1 year of dialysis therapy. An increase in LV mass index was an independent risk factor for the development of subsequent heart failure (hazard ratio, 1.3 per each 20 g/m^2 increase), as was a reduction in fractional shortening (hazard ratio, 1.43 per 5% decrease).

Both LV hypertrophy and LV dilatation will ultimately progress to dilated cardiomyopathy if pressure and volume overload are not appropriately managed. Conversely, there is some evidence of a reduction in LV hypertrophy and possibly in mortality if due attention is paid to these risk factors.

In cardiac failure, ventricular output is maintained at the expense of numerous mechanisms. These include an increase in both end diastolic fiber length and end diastolic volume (i.e through the Frank – Starling mechanism), increased sympathetic activity, enhanced secretion of regulatory hormones (e.g. angiotension II, Arginine vasopressin, vasoactive endothelial hormones, brain and atrial natriuretic peptide) and the presence of ouabain – like substances which, through impairment of the sodium – potassium pump (Na+, K+ ATPase), result in enhanced contractility, albeit at the expense of impaired relaxation.

In a circuit already coping with an elevated LV volume and pressure this can easily result, as previously seen, in a raised pulmonary capillary pressure. The clinical presentation then is one of symptomatic heart failure, with dyspnoea and pulmonary venous congestion, ultimately resulting in acute pulmonary edema.

This end stage clinical manifestation of cardiac disease may result from systolic failure, usually caused by dilated cardiomyopathy or ischemia or both, (or) from diastolic dysfunction in association with LV hypertrophy. The latter is almost as frequent as a cause of recurrent or persistent heart failure in dialysis patients as is dilated cardiomyopathy (Pafrey PS, Harnett JD et al, 1998).



Haemodynamics of HT in CRF

Hypertension is both a cause and complication of renal failure. It is one of the most serious complications of CRF.

It is the major risk factor for CCF, CVA, CAD. After the introduction of recombinant human erythropoietin, anaemia is corrected and aggravation of hypertension occurs, so previously normotensive patients may become hypertensive.

Clinical trials and epidemiologic studies indicate that hypertension is a major risk factor for progressive kidney disease.

Evaluation of subjects screened in a multiple risk factor intervention trial who were monitored over a 16 year period showed that

- Higher the blood pressure was a strong and independent risk factor for the development of ESRD and
- The relative risk for ESRD increased with rising systolic blood pressure independent of diastolic blood pressure.

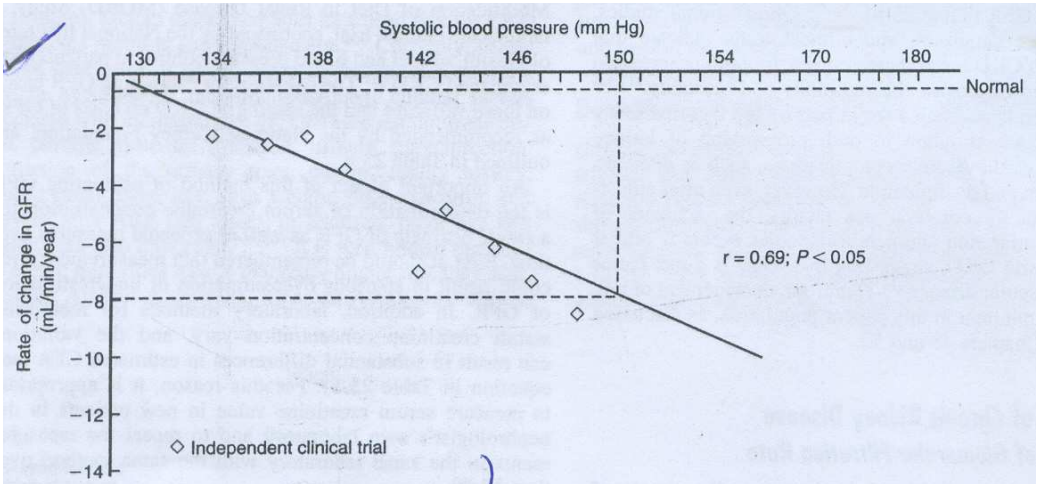
Analysis of National Health and Nutrition Evaluation survey III Data suggests that adequate blood pressure control is achieved in only 11% of patients with hypercreatininemia (serum creatinine > 1.5 mg/dl).

Documentation of blood pressure is essential in the assessment of CRF because this parameter is strongly associated with kidney disease progression and cardio vascular mortality.

The goal blood pressure for patients with CRF is 120 to 139 mm of Hg systolic and 70 to 85 mm of Hg diastolic, depending on the disease type.

The results from nine independent clinical trials five in patients with diabetic nephropathy and four in patients with non diabetic nephropathy indicate that mean rate of decline in GFR is directly associated with level of mean systolic blood pressure (SBP) during the trial.

The rate of decline in GFR in patients with nephropathy even at normal systolic blood pressure, is more than twice that associated with aging in normal individuals.



Absence of hypertension may signify the presence of a salt wasting form of renal diseases such as medullary cystic disease, chronic tubulo interstitial disease, or papillary necrosis and ongoing antihypertensive therapy, volume depletion due to gastrointestinal causes (or) diuretic therapy, or reduced cardiac index.

Since volume overload is the major cause of hypertension in uremia, the normotensive state can often be restored by appropriate (not overzealous) use of salt restriction and natriuretic drugs or ultra filtration in the dialysis setting. Nevertheless, because of hyperreninemia and other disturbances in renal vasoconstrictors and vasodilators, some patients remain hypertensive despite vigorous salt and water restriction and ultrafiltration. Rarely, such patients may develop accelerated (or) malignant hypertension.

Ischemic Cardiovascular disease

CRF at all stages constitutes a major risk factor for ischemic cardiovascular disease, including occlusive coronary heart, cerebro vascular and peripheral vascular diseases. Increased prevalence of coronary heart disease in CRF derives from both traditional (‘’ classic) and CRF- related non traditional risk factors.

The traditional risk factors include hypertension, hypervolemia, dyslipidemia, sympathetic over activity and hyperhomocysteinemia.

The CKD related risks include anaemia, hyperphosphatemia, hyperparathyroidism and a state of “micro inflammation” that can be found at all stages of CRF but is undoubtedly aggravated by dialysis.

The inflammatory state elicits a rise in acute phase reactants such as IL-6 and CRP, which contribute to the coronary occlusive process and are predictors of cardiovascular disease risk. Other abnormalities augment myocardial ischemia. These include reduced myocardial tolerance to ischemia due to left ventricular hypertrophy and microvascular disease.

Nitric oxide is an important mediator for vascular dilatation. Its availability in CRF is decreased because of increased concentrations of asymmetric dimethyl – l – arginine, even at early stages of CRF and also because nitric oxide is scavenged by reactive O_2 species. In addition, coronary arteriolar hypertrophy / hyperplasia limits vasodilatory capacity.

Pericarditis

With the advent of early initiation of renal replacement therapy, pericarditis is now observed more often in underdialysed patients than in predialysis CRF patients. Pericardial pain with respiratory accentuation, accompanied by a friction rub, are the hall marks of uremic pericarditis.

The finding of a multicomponent friction rub strongly supports the diagnosis. Classic electro cardiographic abnormalities include PR interval depression and diffuse ST segment elevation. Pericarditis may be accompanied by the accumulation of pericardial fluid that is readily detected by echocardiography and that sometimes leads to cardiac tamponade. Pericardial fluid in uremic pericarditis is more often hemorrhagic than in viral pericarditis.

Potentially modifiable cardiovascular risk factors in chronic kidney disease - Identified by Cohort studies

	Predialysis	**Renal transplant**			**Dialysis**			
	LVH	**LVH**	**CHF**	**IHD**	**LVH**	**CHF**	**IHD**	**Death**
Anemia	+	+	+	-	+	+	-	+
Hypertension	+	+	+	+	+	+	+	-
Hyperlipidemia	-	-	-	+	-	-	-	-
Smoking	-	-	-	+	-	+	+	+
Hypoalbuminemia	-	-	-	+	-	+	-	+
C-reactive protein	-	-	-	-	-	-	-	+
Homocysteine	-	-	-	-	-	-	-	+
Divalent ion abnormalities	-	-	-	-	-	-	-	+

CHF – Congestive heart failure

IHD – Ischemic heart disease

LVH – Left ventricular hypertrophy

AIM OF THE STUDY

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- To evaluate the cardiac abnormalities in patients of chronic renal failure by echocardiography.

- To assess the prevalence of systolic and diastolic dysfunction in patients with varying degrees of chronic renal failure on conservative management.

- To compare the various cardiac changes in hypertensive CRF patients with normotensive CRF patients.

MATERIALS AND METHODS

MATERIALS AND METHODS

Setting **:** Chronic Renal Failure patients admitted in Madurai Medical College, Government Hospital.

Collaborating Departments: Department of Nephrology, Madurai Medical College Hospital, Madurai.

Department of Cardiology, Madurai Medical College Hospital, Madurai.

Design of Study **:** Prevalence and Comparative study

Period of Study **:** 01-03-2005 to 28-02-2006

Sample Size **:** 120

Selection of Study Subjects :

The study population consists of chronic renal failure patients admitted in this hospital for the period from March 2005 to February 2006. They are basically divided into three groups according to Glomerular Filtration rate calculated by using one of the standard equations, Cockroft – Gault equation and each group consisted of thirty patients. Of the thirty patients in each group, twenty patients were with hypertension and ten patients were without hypertension and not receiving any antihypertensive drugs.

The GFR was calculated by measuring creatinine clearance by using Cockroft Gault equation.

$$= \frac{140\text{-Age} \times \text{Body Weight (Kg)}}{\text{Plasma Creatinine (mg/dl)} \times 72}$$

(Multiply by 0.85 for women)

★ The patients who had GFR between 30 ml/mt and 90 ml/mt are considered as Group I (Mild / moderate CRF).

★ The patients with GFR between 15 ml/mt and 29 ml/mt (severe CRF) are considered as Group II.

★ The patients with GFR below 15 ml/mt (ESRD) are considered as Group III.

★ The age and sex matched healthy controls are considered as Group IV.

Consent :

Informed consent was taken from all subjects participating in this study.

Exclusion Criteria :

The following patients were excluded from the study.

- Patients with AV Fistula
- Patients with diabetes mellitus
- Patients with active or H/O recent infection during last three weeks.
- Patients with H/O coronary artery disease and other cardiac disorders such as valvular heart disease, congenital heart disease etc.
- Patients on haemodialysis.
- Patients on treatment with erythropoietin.
- Patients with H/O smoking and / or alcohol ingestion and all patients with poor echo window.

All subjects underwent various investigations, haemoglobin, total and differential white cell electrolytes count, renal and liver function

tests, urine analysis, lipid profile, Abdomen ultra sound, chest skiagram and 12 lead electro cardiography.

METHOD

Echocardiography

Basic principles

It uses ultrasound to study the disposition and movement of valves and other structures within the heart. It depends on the reflection of ultrasound waves at interfaces between blood and more solid tissues.

In M mode echocardiography the ultrasound is focused into a narrow beam, and the output is a graph against time of the movement, relative to the chest wall of those structures through which the beam passes. Characteristic patterns of movement are produced in for example mitral stenosis and pericardial effusions are easily recognized. Accurate measurements can be made of cardiac dimensions.

In two dimensional real time echo cardiography, the ultrasound beam is swung rapidly back and front over an arc or sector and the resulting information synthesised into a two dimensional map or picture of the positon of the reflecting structures on a television screen.

The picture is the equivalent of a slice through the heart and the structures shown will depend on the position and orientation of the ultrasound crystal.

Because the beam oscillates very rapidly, the ultrasound image accurately produces the movement of structures in the living heart (hence real time). This type of echo cardiography is particularly good at detecting intracardiac masses such as Thrombi (or) tumors or endocarditic vegetations. It is also very useful in sorting out complex structural abnormalities in congenital heart disease.

Doppler cardiography depends on the fact that sound waves reflected from moving objects such as intra cardiac red blood cells, undergo a frequency shift. This can be used to detect the speed and direction of movement of the red cells and thus of the blood in the heart.

Continuous wave Doppler uses a narrow beam of ultrasound in a way analogous to M mode echocardiography.

Pulsed Doppler can sample blood movement at different depths beneath the transducer, and is frequently combined with 2D echocardiography so as to examine blood flow in relation to known intracardiac anatomy. Doppler cardiography is useful in detecting abnormal directions of blood flow eg. Aortic, mitral regurgitations and

estimating pressure gradients which can be calculated from the maximum velocity of flow. Cardiac output can also be measured.

Echocardiographic parameters assessed

All patients underwent two dimensional directed M mode echocardiography performed on Hewlett Packard SIM 7000, using 3.5 MHZ transducer by cardiologists of this hospital.

The left ventricular ejection fraction (EF) and fractional shortening (FS) were taken as measures of LV systolic function. EF was determined by measuring left ventricular volumes in apical 2 chamber view. Left ventricular volumes were measured by Area – length method both in end diastole (LVVd) and in end systole (LVVs).

$$EF = \frac{LVVd - LVVs}{LVVd}$$

The mean EF in normal population is taken as 63.8 $\pm$ 5.1 %. EF was considered decreased if it was < 50%.

Fractional shortening (FS) was determined by measuring left ventricular internal diameter in diastole (LVIDd) and left ventricular internal diameter in systole (LVIDs) by 2D directed M mode echo at the level of papillary muscle.

$$FS = \frac{(LVIDd - LVIDs)}{LVIDd} \times 100$$

Normal reference value in adults for FS is 35±8% FS of ≤ 25% was taken as index of systolic dysfunction.

Diastolic function was determined by ratio of peak early diastole velocity (E)/peak atrial filling velocity (A) of LV i.e. (E/A) measured by spectral doppler LV inflow velocity with sample volume at the level of mitral valve. Normal value of doppler LV diastolic function index was taken as : Peak velocity E (m/sec) : 0.61 m/sec ± 0.14, peak velocity A (m/sec) : 0.48 m/sec ± 0.14 with a normal E/A ratio : 1.40±0.54. LV diastolic dysfunction was considered if E/A velocity was found to be < 1.

Computer analysis of statistical data was done utilizing Epidemiological Information Package (EPI 6) developed by World Health Organisation.

Range, Mean, Standard deviation and ‘p’ values were calculated using this software.Kruskal – Wallis test equivalent to Chi Square test was used to find out the significance of relationship.

RESULTS AND ANALYSIS

RESULTS AND ANALYSIS

Table 1 : Sex

Sex	Group							
	Gr. I (Mild & Moderate CRP)		Gr. II (Severe CRF)		Gr. III (ESRD)		Gr. IV (Controls)	
	No.	%	No.	%	No.	%	No.	%
Male	21	70	20	66.7	19	63.3	21	70
Female	9	30	10	33.3	11	36.7	9	30
Total	30	100	30	100	30	100	30	100

A total number of 60 males and 30 females were selected for the study.

Both sexes were distributed in the same ratio in the study and the control population.

Table 2 : Age

Age	Group							
	Gr. I (Mild & Moderate CRP)		Gr. II (Severe CRF)		Gr. III (ESRD)		Gr. IV (Controls)	
	No.	%	No.	%	No.	%	No.	%
≤ 30	1	3.3	6	20	3	10	1	3.3
31-40	17		13		14		15	50
41-50	10		5		12		11	
> 50	2		6	20	1		3	
Total	30	100	30	100	30	100	30	100
Range	30-55		28-58		29-52		28-58	
Mean	39.5		38.9		39.4		40.3	
S.D.	6.7		9.3		6.4		7.1	

'p' = 0.1223 (Not significant)

Majority of the patients in both study and control group lies between 31 and 40 years. Almost same age group of patients were selected in both groups and there is no statistically significant difference in the age composition.

Table 3 Weight

Weight	Group			
	Gr. I (Mild & Moderate CRP)	Gr. II (Severe CRF)	Gr. III (ESRD)	Gr. IV (Controls)
Mean	61.1	62.0	56.0	59.2
S.D.	6.1	5.0	6.8	5.4
'p'	0.004			

When compared to Group I patients, the average body weight is less in Group III patients. The difference is significant also.

Table 4 Hb%

Hb%	Group			
	Gr. I (Mild & Moderate CRP)	Gr. II (Severe CRF)	Gr. III (ESRD)	Gr. IV (Controls)
Mean	8.82	7.18	6.07	11.92
S.D.	0.74	0.82	0.6	0.9
'p'	0.0001			

When compared to Group I patients, the average Hb level is less in Group II and Group III patients and it is statistically significant. As expected, the mean Hb level showed a progressive decline with the severity of renal failure.

Table 5 Urea

Urea	Group			
	Gr. I (Mild & Moderate CRP)	Gr. II (Severe CRF)	Gr. III (ESRD)	Gr. IV (Controls)
Mean	69.1	132.9	165.6	20.1
S.D.	10.3	12.3	18.5	2.9
‘p’	0.0001			

Table 6 Creatinine

Creatinine	Group			
	Gr. I (Mild & Moderate CRP)	Gr. II (Severe CRF)	Gr. III (ESRD)	Gr. IV (Controls)
Mean	2.33	4.66	7.0	0.7
S.D.	0.43	0.53	0.96	0.22
‘p’	0.0001			

As evident from Table 5 and Table 6, Blood urea and creatinine levels showed a progressive increase in their levels with increase in severity of renal failure which was statistically significant when compared to controls. ('p' < 0.05)

Table 7 Echocardiographic parameters in 4 groups

(in cm)	Group			
	Gr. I (Mild & Moderate CRP)	Gr. II (Severe CRF)	Gr. III (ESRD)	Gr. IV (Controls)
LVIDd	3.7±0.8	4.2±0.4	4.8±0.6	3.3±0.4
LVIDs	2.6±0.7	2.9±0.6	3.3±0.4	2.2±0.3
IVSd	1.3±0.3	1.4±0.2	1.6±0.4	1.1±0.2
IVSs	1.4±0.4	1.5±0.2	1.8±0.5	1.3±0.1
PWd	1.1±0.2	1.2±0.2	1.3±0.2	1.0±0.1
LVVd (cubic ml)	78.6±24.5	88.1±26.2	94±31.2	72.7±9.3
LVVs(Cube ml)	33.4±15.4	38.2±15.5	43.2±16.2	28.5±5.4

It is seen from the above Table, that the mean left ventricular internal diameter in diastolic (LVIDd) and systolic (LVIDs) as well as the mean left ventricular volume in both systolic (LVVd) and diastolic (LVVd) were higher in the patients with mild/ moderate CRF and severe CRF groups than the control group.

Table 8 : EF

EF	Group							
	Gr. I (Mild & Moderate CRP)		Gr. II (Severe CRF)		Gr. III (ESRD)		Gr. IV (Controls)	
	No.	%	No.	%	No.	%	No.	%
Normal (> 50%)	29	96.7	24	80	20	66.7	30	100
Decreased (<50%)	1	3.3	6	20	10	33.3	-	-
Total	30	100	30	100	30	100	30	100
Range	47.2-63.8		42.8-61.1		36.7-58.5		58.9-68.9	
Mean	59.5		56.3		51		63.1	
S.D.	3.0		5.9		8.6		3.6	

Group I versus Group IV – p = 0.5
Group II versus Group IV – p = 0.01186
Group III versus Group IV – p = 0.0018
Group I versus Group III – p = 0.0076
Group II versus Group III – p = 0.3811
Group I versus Group II – p = 0.0514

Table 8 shows indices of Left Ventricular (LV) systolic function in the four groups. As evident, LV Ejection Fraction showed a progressive decline with increase in severity of renal failure which was statistically significant ('p'<0.05) when Group II and Group III patients are compared to controls. Further analysis showed that whereas in patients with mild / moderate CRF only 1 out of 30 (3.3%) patients showed low EF (<50%), 6 out of 30 (20%) patients with severe CRF and 10 out of 30 (33.3%) with ESRD had evidence of low EF.

Table 9 : Fractional Shortening (FS)

FS	Group							
	Gr. I (Mild & Moderate CRP)		Gr. II (Severe CRF)		Gr. III (ESRD)		Gr. IV (Controls)	
	No.	%	No.	%	No.	%	No.	%
Normal (> 25%)	29	96.7	17	56.7	14	46.7	30	100
Decreased (<25%)	1	3.3	13	43.3	16	53.3	-	-
Total	30	100	30	100	30	100	30	100
Range	24.9-36.1		22-34.2		18.4-30		29.9-37.1	
Mean	32.5		27.4		23.9		33.3	
S.D.	2.6		4.3		4.5		2.6	

Group I versus Group IV – p = 0.5
Group II versus Group IV – p = 0.0002
Group III versus Group IV – p = 0.0001
Group I versus Group III – p = 0.0001
Group II versus Group III – p = 0.6053
Group I versus Group II – p = 0.0008

As evident from Table 9, LV fractional shortening showed a progressive decline with increase in severity of renal failure. There is no statistically significant difference ('p'>0.05) between Group II and Group III.. Detailed analysis revealed that 1 out of 30 (3.3%) patients with mild / moderate CRF, 13 out of 30 (43.3%) patients with severe CRF and 16 out of 30 (53.3%) with ESRD had impaired fractional shortening (i.e., $\leq$ 25%)

Table 10 E/A Ratio

E/A Ratio	Group							
	Gr. I (Mild & Moderate CRP)		Gr. II (Severe CRF)		Gr. III (ESRD)		Gr. IV (Controls)	
	No.	%	No.	%	No.	%	No.	%
Normal (≥ 1)	24	80	18	60	11	36.7	30	100
Decreased(<1)	6	20	11	36.7	17	56.7	-	-
Pseudonormalisation	-	-	1	3.3	2	6.7	-	-
Total	30	100	30	100	30	100	30	100
Range	0.92-1.51		0.93-1.51		0.92-1.68		0.92-1.73	
Mean	1.115		1.101		1.115		1.483	
Standard deviation	0.17		0.18		0.25		0.12	

Group I versus Group IV (Controls)– p = 0.0119
Group II versus Group IV – p = 0.1211
Group III versus Group IV – p = 0.0001
Group I versus Group III – p = 0.0017
Group II versus Group III – p = 0.1211
Group I versus Group II – p = 0.159

Table 10 shows diastolic functions in the four groups. As evident, the mean E/A was significantly lower in patients with mild / moderate CRF, severe CRF and ESRD when compared to controls. In mild / moderate CRF group 6 out of 30 patients (20%),in severe CRF 11 out of 30 (36.7%) and in ESRD 17 out of 30 (56.7%) had diastolic dysfunction (E/A ≤1) .Thus the Group III patients had significantly impaired diastolic function when compared to Group I patients.

Table 11 Left Ventricular Hypertrophy (LVH)

LVH	Group							
	Gr. I (Mild Moderate CRP)		Gr. II (Severe CRF)		Gr. III (ESRD)		Gr. IV (Controls)	
	No.	%	No.	%	No.	%	No.	%
ConcentricHypertrophy	4	13.3	9	30	8	26.7	-	-
Eccentric hypertrophy	-	-	3	10	6	20	-	-
Total	4	13.3	12	40	14	43.3	-	-
Range	118-238		145-275		147-324		110-217	
Mean	167.9		209		234.1		176.4	
Standard deviation	32.9		42.7		63.7		25.6	

Group I versus Group IV (Controls) – p = 0.0562
Group II versus Group IV (Controls) – p = 0.0004
Group III versus Group IV (Controls) – p = 0.0002
Group I versus Group III – p = 0.0219
Group II versus Group III – p = 1.00
Group I versus Group II – p = 0.041

Table 11 shows the patients of LVH according to the severity of renal failure. The number of patients with concentric LVH among Group I patients was 4 (13.3%). Detailed analysis revealed that 4 out of 30 (13.3%) patients with mild / moderate CRF, 9 out of 30 (30 %) patients with severe CRF and 8 out of 30 (26.7%) with ESRD had concentric LVH whereas Nil patients in Group I, 3 out of 30 (10%) patients in Group II and 6 out of 30 (20%) in Group III had eccentric LVH (dilation of LV).

Table 12 : Hypertension and EF

EF	Group I		Group II		Group III		Total RF	
	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)
Normal	19	10	15	9	13	7	47	26
Decreased	1	-	5	1	7	3	13	4

p=0.0001

In normotensive CRF patients, Nil patients with mild to moderate CRF, 1 out of 10 (10 %) patients with severe CRF and 3 out of 10 (30%) with ESRD had low Ejection Fraction (systolic dysfunction). Whereas in hypertensive CRF patients, 1 out of 20 (5%) patients with mild to moderate CRF, 5 out of 20 (25 %) patients with severe CRF and 7 out of 20 (35%) with ESRD had low Ejection Fraction.

Table 13 : Hypertension and FS

FS	Group I		Group II		Group III		Total RF	
	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)
Normal	19	10	9	8	8	6	36	24
Decreased	1	-	11	2	12	4	24	6

p=0.0001

In normotensive CRF patients, Nil patients with mild to moderate CRF, 2 out of 10 (20 %) patients with severe CRF and 4 out of 10 (40%) with ESRD had low Fractional Shortening. Whereas in hypertensive CRF patients, 1 out of 20 (5%) patients with mild to moderate CRF, 11 out of 20 (55 %) patients with severe CRF and 12 out of 20 (60%) with ESRD had low fractional shortening.

Table 14 Hypertension and E/A

E/A Ratio	Group I		Group II		Group III		Total RF	
	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)
Normal	16	8	10	8	7	4	33	20
Decreased	4	2	9	2	11	6	24	10
Pseudo normalisation	-	-	1	-	2	-	3	-

p=0.6703

In normotensive CRF patients, 2 / 10(20%) patients with mild to moderate CRF, 2 / 10 (20 %) patients with severe CRF and 6 / 10 (60%) with ESRD had E/A slope < 1 (diastolic dysfunction).

In hypertensive CRF patients, 4 / 20 (20 %) patients with mild to moderate CRF, 10 / 20 (50 %) patients with severe CRF and 13 / 20 (65%) with ESRD including patients with pseudonormal pattern had diastolic dysfunction.

Table 15 Hypertension and LVH

LVH	Group I		Group II		Group III		Total RF	
	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)	A (with HT)	B (Without HT)
Positive	4	-	11	1	10	3	25	4
Negative	16	10	9	9	10	7	35	26

p=0.0001

In normotensive CRF patients, nil patients with mild to moderate CRF, 1/10 (10%) patient with severe CRF and 3/10 (30%) patients with ESRD had LVH.

In hypertensive CRF patients, 4/20 (20%) patients with mild to moderate CRF, 11/20 (55%) patients with severe CRF and 11/20 (55%) patients with ESRD had LVH.

DISCUSSION

DISCUSSION

Premature cardiovascular disease is a significant cause of morbidity and mortality among patients with CRF. Various diagnostic modalities, both invasive and non invasive such as electrocardiography, echocardiography, and radionucleide scans are utilised for diagnosing left ventricular hypertrophy and dysfunction. Echocardiography provides an excellent non invasive method to delineate details of anatomy of cardiac cavity, wall dimensions, and wall movements.

The study population consists of chronic renal failure patients admitted in this hospital for the period from March 2005 to February 2006. They are basically divided into three groups according to Glomerular Filtration rate calculated by using one of the standard equations, Cockroft – Gault equation and each group consisted of thirty patients. In my study, of the thirty patients in each group, twenty patients were with hypertension and ten patients were without hypertension and not receiving any antihypertensive drugs.

The patients who had GFR between 30 ml/mt and 90 ml/mt are considered as Group I (Mild / moderate CRF).The patients with GFR

between 15 ml/mt and 29 ml/mt (severe CRF) are considered as Group II.The patients with GFR below 15 ml/mt (ESRD) are considered as Group III.The age and sex matched healthy controls are considered as Group IV.

Comparative Analysis

The study population in S. Agarwal et al study, consisted of three groups: group A (n=30) – age and sex matched healthy controls; group B (n=30) – patients with mild to moderate CRF (S. creatinine 1.5 – 6.0mg/dl): and group C (n=30) – patients with advanced CRF (S. creatinine > 6.0mg/dl).

In a study conducted by R.V. Prasad et al, Kanpur 35 cases were divided in to three groups. Group A consisted of normal healthy controls. Group B and Group C were formed by hypertensive CRF patients and normotensive CRF patients respectively.

Raj DS, D' Mellos conducted a study in which the echo candiographic assessment was done in 38 chronic renal failure patients on conservative management, 35 patients on hemodialysis and 36 matched controls.

In our study, the patients were grouped according to GFR whereas In S. Agarwal et al study, the patients were grouped on the

basis of serum creatinine level. By using GFR, the patients can be appropriately classified into various degrees of renal failure rather than using only creatinine values.

In our study, among 90 patients with various degrees of renal failure, 60 patients were male and 30 patients were females. So the male to female ratio is 2:1.

In S. Agarwal et al study, among 60 patients with CRF, 34 patients were male and 26 patients were females. Male patients are more prone to chronic renal failure than women.

In Agarwal et al study, the age incidence was common among 31-40 years. In our study also, the observation was similar.

In the western population, the age incidence is more in 51-60 years (Barry N. Brenner). So in our Indian population, the younger age group are more affected. Because the bacterial infections like streptococcus and viral infection are more in our country than western country where diabetes mellitus and hypertension in old age group constitute the majority of chronic renal failure population.

The dry body weight decreases as the severity of renal failure increases because they suffer from chronic acidosis, malnutrition and anorexia. It has been observed in our study population.

As expected mean Hb level showed a progressive decline with the severity of renal failure. The results are similar to that of the study made by Dr. S. Agarwal, Dr. Rajpal, New Delhi 2003.

The mean Hb values in our study are 8.8g/dl in Group1 (mild/moderate CRF), 7.1g/dl in Group II (severe CRF), and 6.0g/dl in Group III (ESRD) patients. In Agarwal et al study, the mean Hb values were 13.2g/dl in mild/moderate CRF and 7.3g/dl in severe CRF.

Mean values of Echocardiographic parameters in severe CRF

Echo – parameters	Agarwal et al study	Present study
LVIDd (cm)	4.7	4.8
LVIDs (cm)	3.1	3.3
LVVd (cubic ml)	92	94
LVVs (cubic ml)	41	43.2

From this study, it is evident that as severity of renal failure increases, the mean left ventricular internal diameter (LVIDd) and systole (LVDs) as well as the mean left ventricular volume in both systole (LVVs) and diastole (LVVd) becomes more.

Most of the studies made in Western population and Indian patients were based on serum creatinine levels in classifying the severity of CRF patients. (Greaves et al (1994), Raj et al (1997), Ayus et al (1981) and Dr. Agarwal and Rajpal (2003), New Delhi). In my study the patients were classified on the basis of creatinine clearance which was calculated by using Cockroft Gault equation.

In the present study, the mean ejection fraction in patients with mild/moderate CRF and severe CRF groups showed a downward trend. Among patients in the mild / moderate CRF group only one patient had LVEF < 50%, while in advanced renal failure states (Group II and III), 16/60 (27%) patients had LVEF which was significantly different from controls, as well as mild/ moderate CRF population.

In S. Agarwal et al study, among patients in mild/moderate CRF only one patient had ejection fraction less than 50% (3.3%). while in severe CRF, 9/30 (30%) patients had low ejection fraction. The

tendency of decrease in EF values with increase in severity of renal failure are similar in both study groups.

On analyzing the parameter, fractional shortening (FS), the percentage of Group I patients having FS less than 25% is 3.3% which is similar to that of patients with EF less than 50% whereas in Group II patients and III patients, more number of patients have low FS when compared to patients with low EF.

Group II patients with low EF and low FS are 20% and 43% respectively. Group III patients with low EF and low FS are 33% and 53% respectively.

It is because when the ventricle is stressed by a haemodynamic overload, it first uses its compensatory mechanism to maintain normal mechanical performance and the ejection indices (eg. Ejection fraction) within normal limits. It is only when all the compensatory mechanisms in the form of operation of Frank Starling's mechanism, development of hypertrophy, and endogenous adrenergic stimulation have been maximally used, there is decline in ejection phase indices.

In Agarwal et al study, the number of patients showing low FS (< 25%) were 7 patients (23%) in mild to moderate CRF and in severe CRF, there were 5 patients (16.6%).

Among these two parameters (EF and FS), for assessing systolic function the ejection fraction is more reliable when compared to fractional shortening because FS assesses the status of basal chamber only; it may falsely be normal, depressed (or) increased in segmentally depressed left ventricle (Feigenbaum H ; Braunwald EEd Heart disease, 1992).

Left ventricular diastolic dysfunction develops in three phases;

1. Inversion of ratio of peak early to peak atrial velocity – curve with reduced early ventricular filling, due to reduced ventricular relaxation,
2. Pseudonormalisation of E/A flow pattern, following increased atrial and ventricular filling pressures and decreased ventricular relaxation.
3. Development of a restrictive pattern with various degrees of early atrial flow velocity involvement.

Thus it may be difficult to interpret whether a normal E/A flow pattern is associated with normal heart function or might in fact be related to progressive diastolic heart dysfunction, with increased filling pressure, unless the pulmonary venous flow is analysed (Schroeder AP, Neilsen CB et al, 1997).

In the present study, the mean E/A ratio in control group was 1.48. It was 1.11 in mild/ moderate CRF patients, 1.101 in severe CRF and 1.115 in ESRD patients (the difference being statistically significant). The prevalence of diastolic dysfunction in the present study was found to be 20% (6 patients) in mild/moderate CRF, 36.7% (11 patients) in severe CRF group and 56.7% (17 patients) in ESRD group.

The prevalence of systolic dysfunction as evidenced by low EF was found to be 3.3% (1 patient) in mild/ moderate CRF, 20% (6 patients) in severe CRF and 33% (10 patients) in ESRD group.

Thus in comparison to systolic function, diastolic function was deranged in more number of patients suggesting that diastolic function is first to appear in patients with chronic renal failure.

All the patients with E/A ratio more than 1 were further studied by pulmonary venous flow pattern during echocardiography one patient from Group II and two patients from Group III with E/A ratio > 1 were found to have large atrial reversal in pulmonary venous flow pattern (Pseudo normalization of E wave).

In S. Agarwal et al study, the mean E/A ratio in control group was 1.4. It was 0.92 in mild/moderate CRF patients and 0.96 in severe CRF group (the difference being statistically significant). Similarly, Schroeder et al (1992) had found E/A ratio of velocity time indices to be 1.45 in patients with mild renal impairment as compared to 1.99 in controls. Also, in a study of ESRD patients by Virtanen et al (1998), mean E/A was 1.5 + 0.5. London et al (1993) reported a significant reduction in E/A ratio in haemodialysis patients as compared to controls. The prevalence of diastolic dysfunction in S. Agarwal et al study was found to be 66.6% (20patients) in mild/moderate CRF group and 53.2% (16 patients) in severe CRF group.

Among the various factors that constitute to diastolic and systolic dysfunction, uncontrolled hypertension and anaemia which are usually present in CRF, play a significant role. This has been observed in our study.

Left ventricular hypertrophy (LVH) is the single strongest independent predictor of adverse cardiovascular events. LVH is a major echocardiographic finding in uremic patients.

In the present study, we found that LVMI (LV Mass Index) showed a progressive rise with increase in severity of renal failure. This is in concordance with the study done by Greaves and co workers and Dr. S. Agarwal and co workers who also found a similar trend of LVMI in patients of CRF.

A study done by Harnett et al revealed that a session of hemodialysis significantly decreased LVMI in CRF patients.

In the present study, 4 patients (13%) in mild/ moderate CRF group, 12 patients (40%) in severe CRF and 13 patients (43%) in ESRD had LVH.

It was also observed that concentric LVH is far more common than eccentric LVH (LV dilatation) in CRF population.

As renal failure advances, the number of patients developing eccentric LVH increases as observed in our study.

In P. Dangri and Agarwal et al study, 40% (12/30) of patients in mild/moderate CRF group and 96% (29/30) patients in severe CRF group had LVH. Concentric LVH was found in 11 out of 12 patients in mild/moderate CRF and 26 out of 29 patients in severe CRF. Eccentric LVH (LV dilatation) was found in one patient in mild/moderate CRF and 3 patients in severe CRF. In our study and P. Dangri et al study, concentric LVH was far more common than eccentric hypertrophy.

In comparing various measurements in hypertensive CRF patients (20 patients in each group) with normotensive CRF patients (10 patients in each group) all the parameters were significantly deranged.

LVH was observed in 13% of patients of normotensive CRF and it was seen in 43% of patients with hypertensive CRF. There was a direct correlation between LVMI and severity of CRF in both normotensive and hypertensive groups.

The systolic dysfunction was observed in 13% of patients with normotensive CRF whereas in hypertensive CRF patients, the systolic dysfunction was observed in 21% of patients.

The diastolic dysfunction was observed in 20% of patients with normotensive CRF whereas in hypertensive CRF patients, the diastolic dysfunction was noted in 40% of patients. So it can be understood that even in normotensive CRF patients, the diastolic dysfunction is more when compared to systolic dysfunction.

In R.V. Prasad et al study, among normotensive CRF patients 30% of patients had systolic dysfunction where as, among hypertensive CRF patients about 66% of patients were found to have systolic dysfunction. ($p < 0.05$). The same study showed diastolic dysfunction in 86.6% in hypertensive CRF patients and 50% in normotensive CRF patients ($p < 0.05$)

Pericardial effusion was present in 5 patients in Group II (16%) and 8 patients in Group III (26%). Most of the patients had mild to moderate pericardial effusion and none of them had cardiac tamponade. Pericardial thickening was present in 2 patients with ESRD (Group III).

Pericardial involvement was found in 5.5% (5/90) of patients with CRF clinically but echo showed mild to moderate effusion in 14% (13/90) of patients. It directly correlated with severity of renal failure.

In R.V. Prasad et al study pericardial involvement was found in 8% of patients clinically but echo showed effusion in 36% ($p < 0.05$).

Mitral regurgitation was observed in 3 (10%) patients in Group II and 5 patients (17%) in Group II . They had Grade I to Grade II MR. They were mainly observed in patients with eccentric hypertrophy (LV dilatation).

CONCLUSION

CONCLUSION

The following conclusions were derived from this study.

1. Majority of the patients in all groups of patients were in the age group of 31-40 years.

2. The mean Hb level showed a progressive decline with the severity of renal failure.

3. The average body weight is less in Group III (ESRD) patients.

4. Blood urea and creatinine levels showed a progressive increase in their levels with increase in severity of renal failure.

5. The echocardiographic parameters such as LVIDd, LVIDs and LVVd and LVVd were higher in the patients with mild / moderate CRF and severe CRF groups than the control group.

6. 3.3% of patients with mild to moderate CRF, 20% of patients with severe CRF and 33.3% of patients with ESRD had systolic dysfunction.

7. 20% of patients with mild to moderate CRF, 36% of patients with severe CRF and 56% of patients with ESRD had diastolic dysfunction.

8. It can be understood that patients with chronic renal failure have higher prevalence of diastolic and systolic dysfunction and diastolic dysfunction appears to occur earlier than systolic dysfunction.

9. 13.3% of patients with mild / moderate CRF, 40% of patients with severe CRF and 43% of patients had left ventricular hypertrophy.

10. In comparison with normotensive CRF patients, all the echocardiographic parameters were significantly deranged in hypertensive CRF Patients.

11. Clinically there was no valvular involvement but echo showed regurgitant lesions especially of mitral valve in a significant number of cases.

12. Calcification in the cardiac valves was not made out in any of the patients in our study and regional wall motion abnormality was not found in any of the patients and thrombus in the dilated ventricle was not found in any of the patients.

Thus, echocardiography permits rapid and non invasive detection of cardiovascular complication in CRF, some of which remains unrecognized or even unsuspected during clinical examination, chest x-ray or ECG.

SUMMARY

SUMMARY

Patients with chronic kidney disease (CRF) most often present with non specific complaints (or) are asymptomatic and are suspected to have the disease because of abnormal blood or urine findings. So it is important to do complete evaluation for establishing the diagnosis, cause of CRF and various hemodynamic abnormalities associated with CRF.

In particular, evaluation of cardio vascular risk factors is critical because of the high rate of cardio vascular complications in CRF.

Echocardiography is one of the non invasive and sensitive tests for evaluating various cardiac changes in CRF patients at an early stage.

In the cardiovascular system, left ventricular hypertrophy and left ventricular diastolic and systolic dysfunction are the most frequent findings. Cardiac disease frequently predates the start of dialysis and cardiac dysfunction is the major impediment to rehabilitation. So echocardiography should be performed early in the course of CRF and may be valuable in the monitoring of therapy of

these patients with modalities such as blood pressure control, correction of anemia, treatment of dyslipidemia and use of aspirin and statins in selected cases.

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PROFORMA

PROFORMA

Name: IP No:

Age/Sex: Body Weight: (kg)

Admitted for: duration Creatinine clearance ml/mt/1.73m^2

Sweating of legs Y/N Past History

Puffiness of face Y/N HT

Scanty micturition Y/N DM

Easy fatigability Y/N PT

Anorexia Y/N IHD

Nausea, vomiting Y/N Others

Breathlessness Y/N

Family History

GENERAL EXAMINATION ANAEMIA +/- Pedaledeme +/- Dyspnoea +/-

Vital signs **PR** /Mt **BP** /mm of Hg **RR** /mt **Temp**

CVS

RS

ABD

CNS

INVESTIGATIONS

Urine Alb Sug Dep

Spot PCR (Pr : Cr)

Blood – Tc DC-P: L: M: E: Hb%

Blood Sugar Urea Creatinine
Serum Calcium Phosphorous **Electrolytes** Na^+
K^+
$C1^-$
HCO_3^-)

Lipid profile

LFT

ECG

USG abd

Echo

Echocardiographic findings (Proforma)

IVSd (cm)

LVIDd (cm)

PWd(cm)

IVSs(cm)

LVIDs (cm)

PWa(cm)

LVVs(Cubic ml)

LVVd(Cubic ml)

EF

FS

LVVM (g)

Pericardial effusion

Pericardial thickening

DOPPLER STUDY

1. Mitral Flow
   E
   A
   E/A Ratio
   MR-
2. Pul.Venous flow

3. Tricuspid Flow

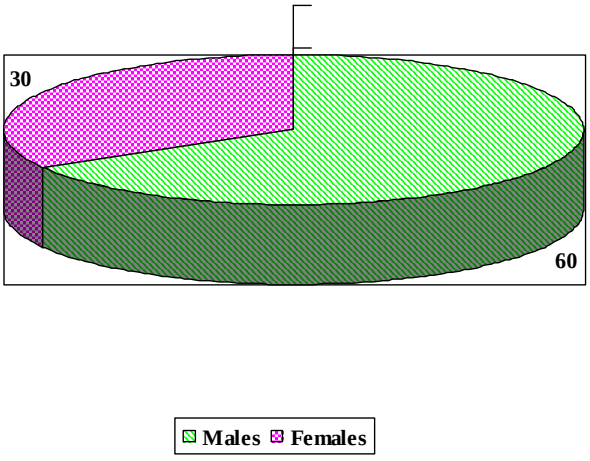
4. Other abnormalities

Thrombus
Pul HT

Evidence of tamponade/
Constriction

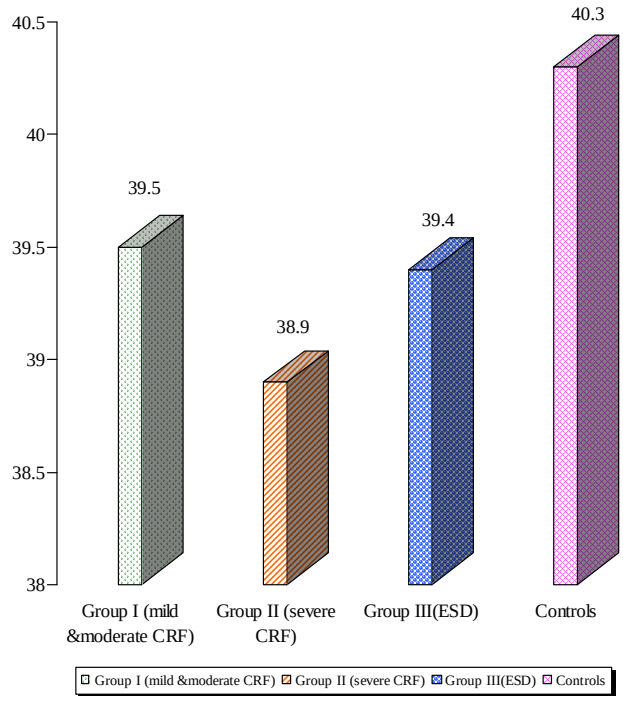
Impression:

Sex

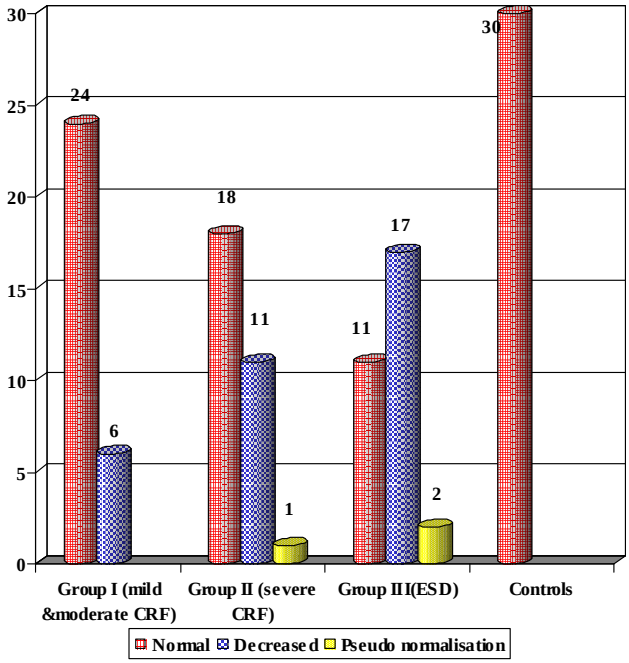


GRAPHS

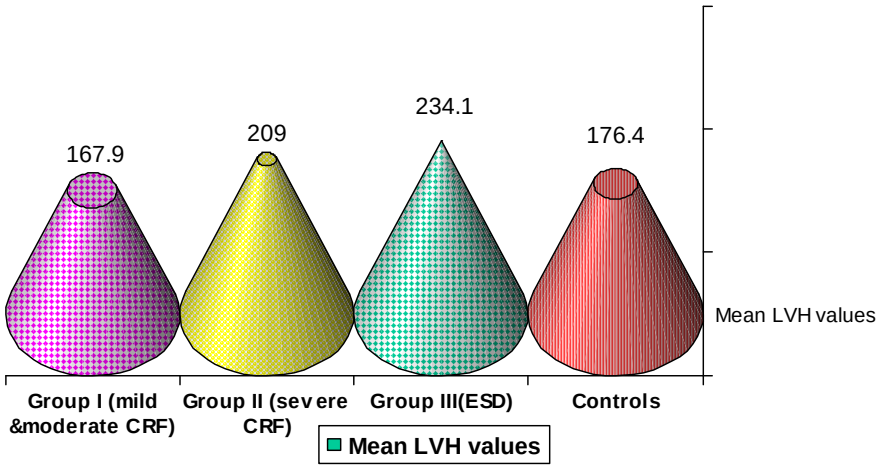
Mean age of the four groups



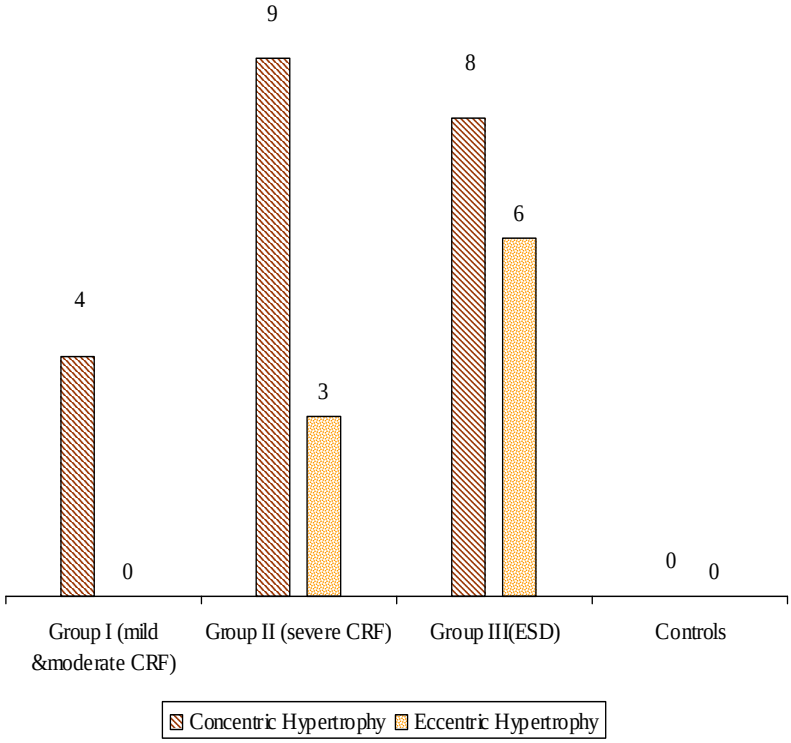
E/A Ratio for the four Groups



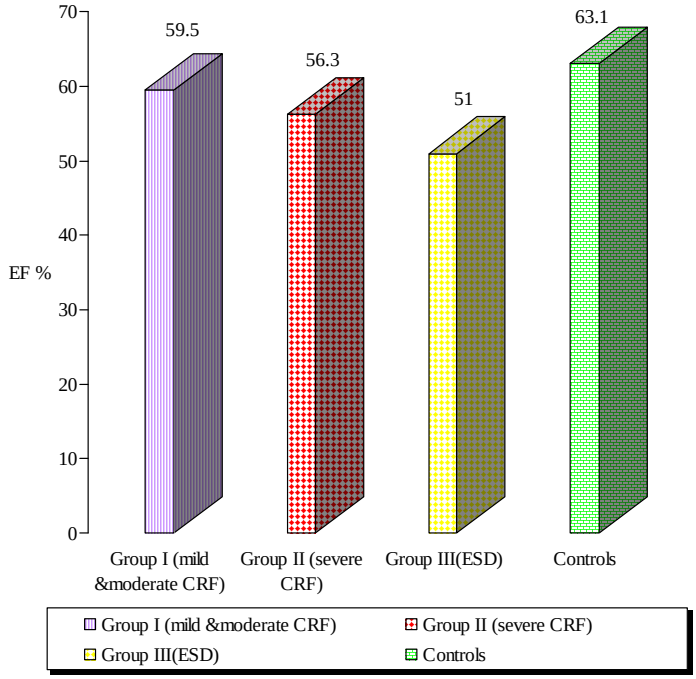
Mean LVH values in the four Groups



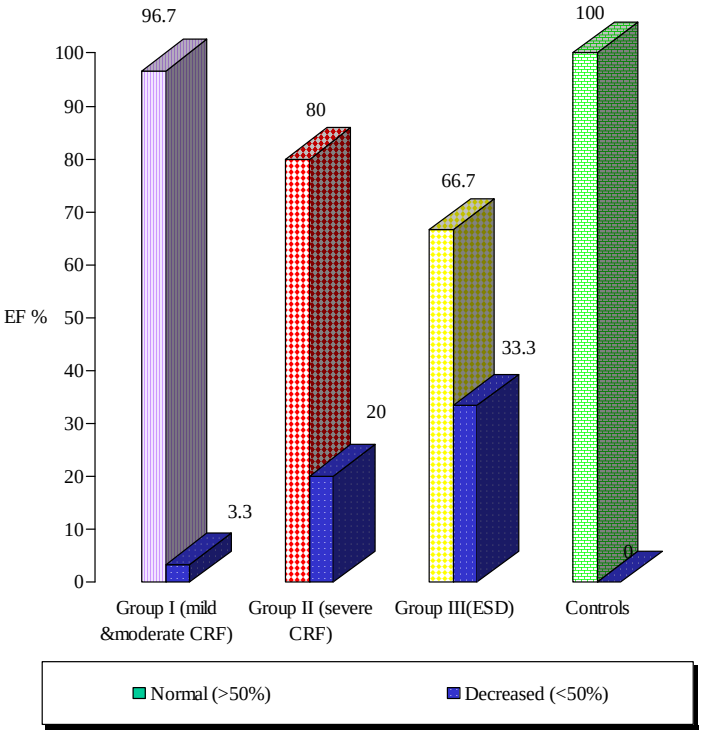
No. of patients with LVH



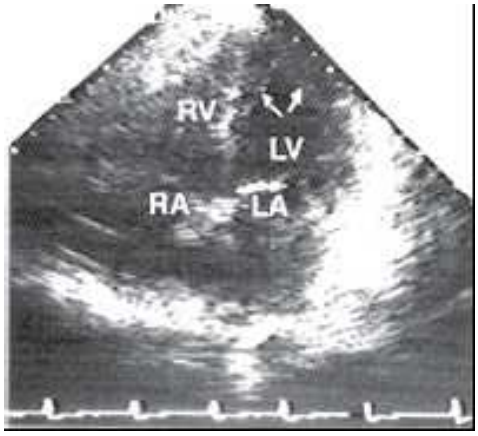
Mean EF values in the four Groups



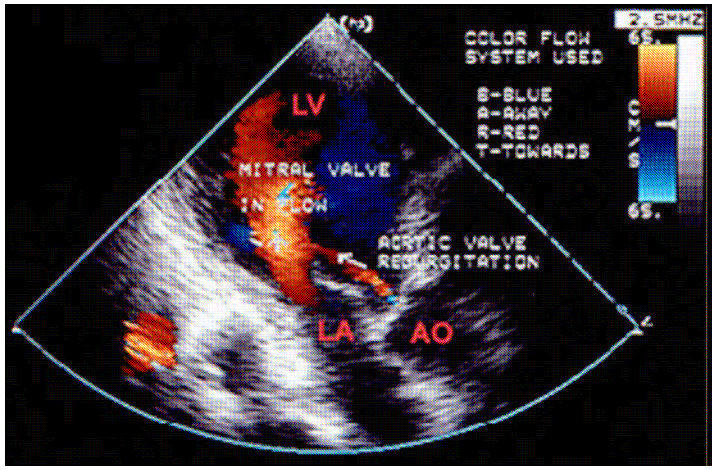
Percentage of patients with normal and low EF values



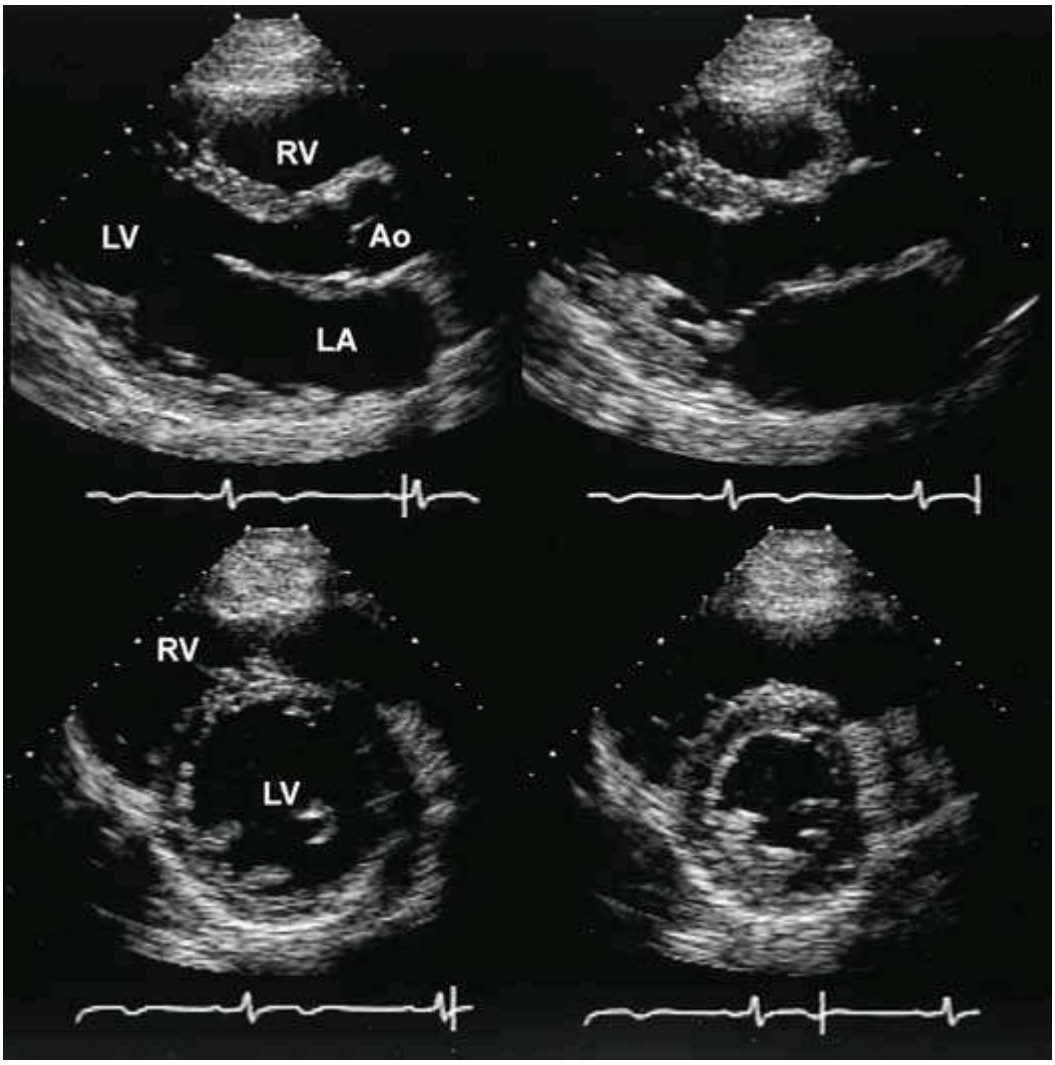
PICTURES



Two-dimensional (2-D) Echo – Normal study

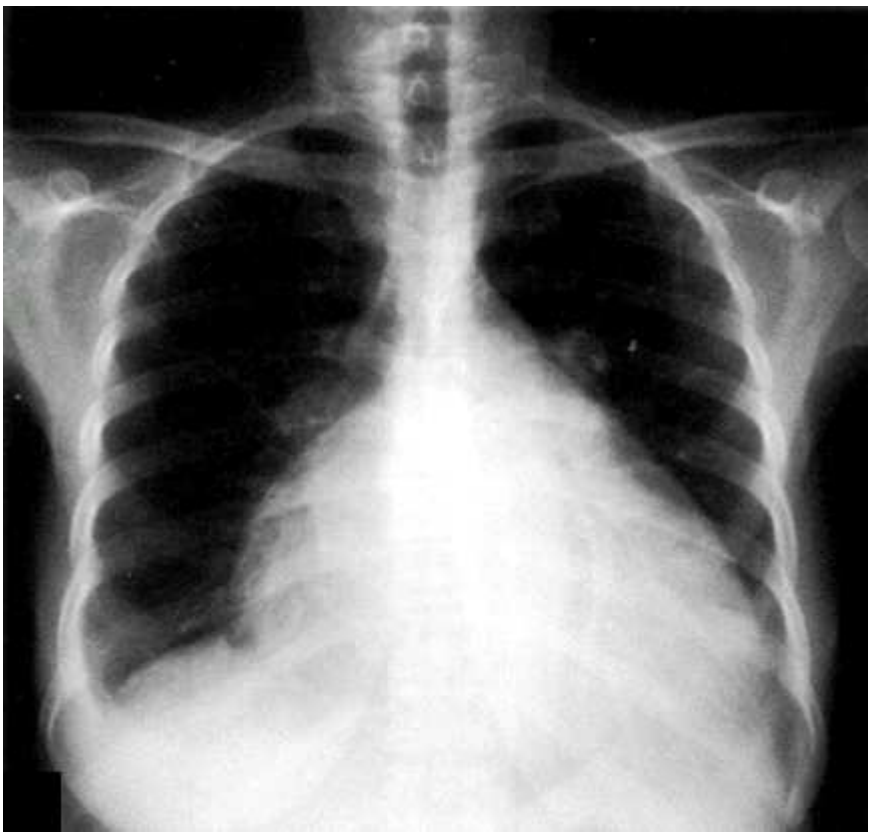


Colour Doppler Echo – Normal study

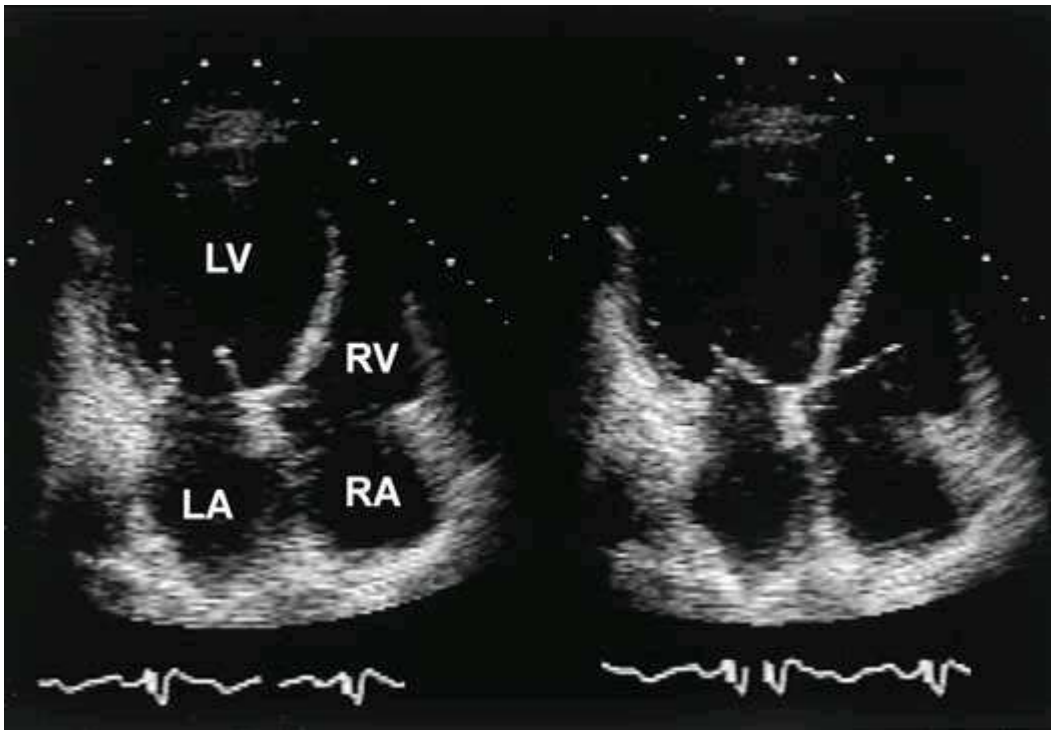


2D Echo - normal heart. *Upper*: Parasternal long axis view during diastole (*left*) and systole (*right*). *Lower*: Parasternal short axis view during diastole (*left*) and systole (*right*)

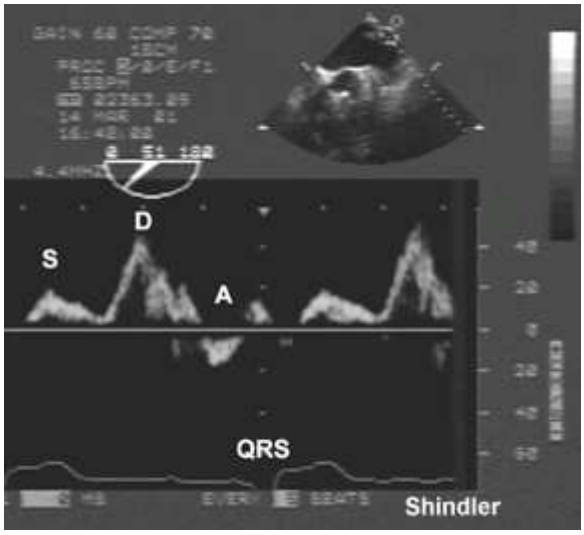
CXR PA view showing Cardiomegaly with pulmonary congestion in a pt with CRF with CCF



Chest X-ray PA View – Pericardial effusion & Bilateral pleural effusion in a case of severe CRF

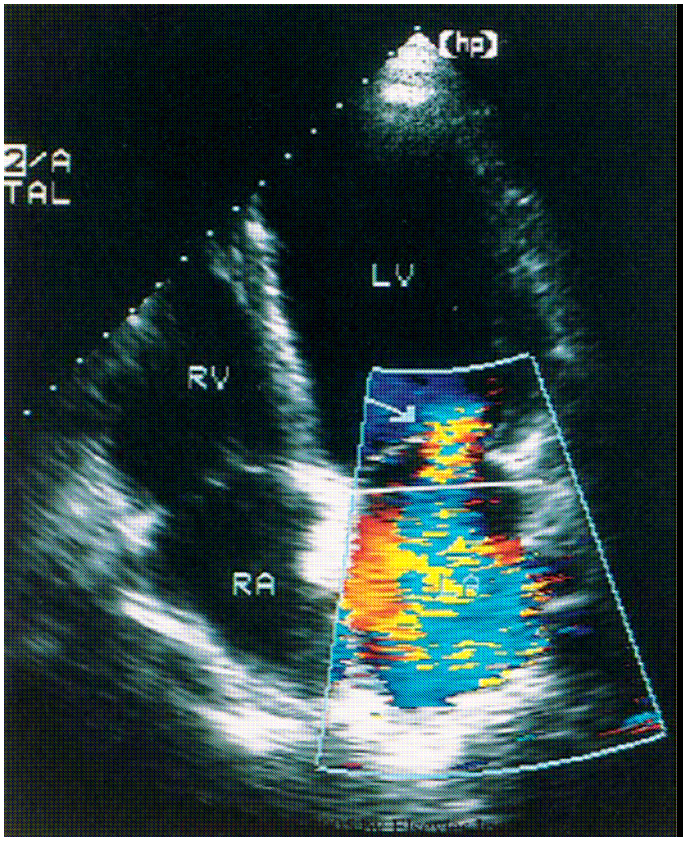


Echo picture of a case of ESRD – Severe cardiac dysfunction

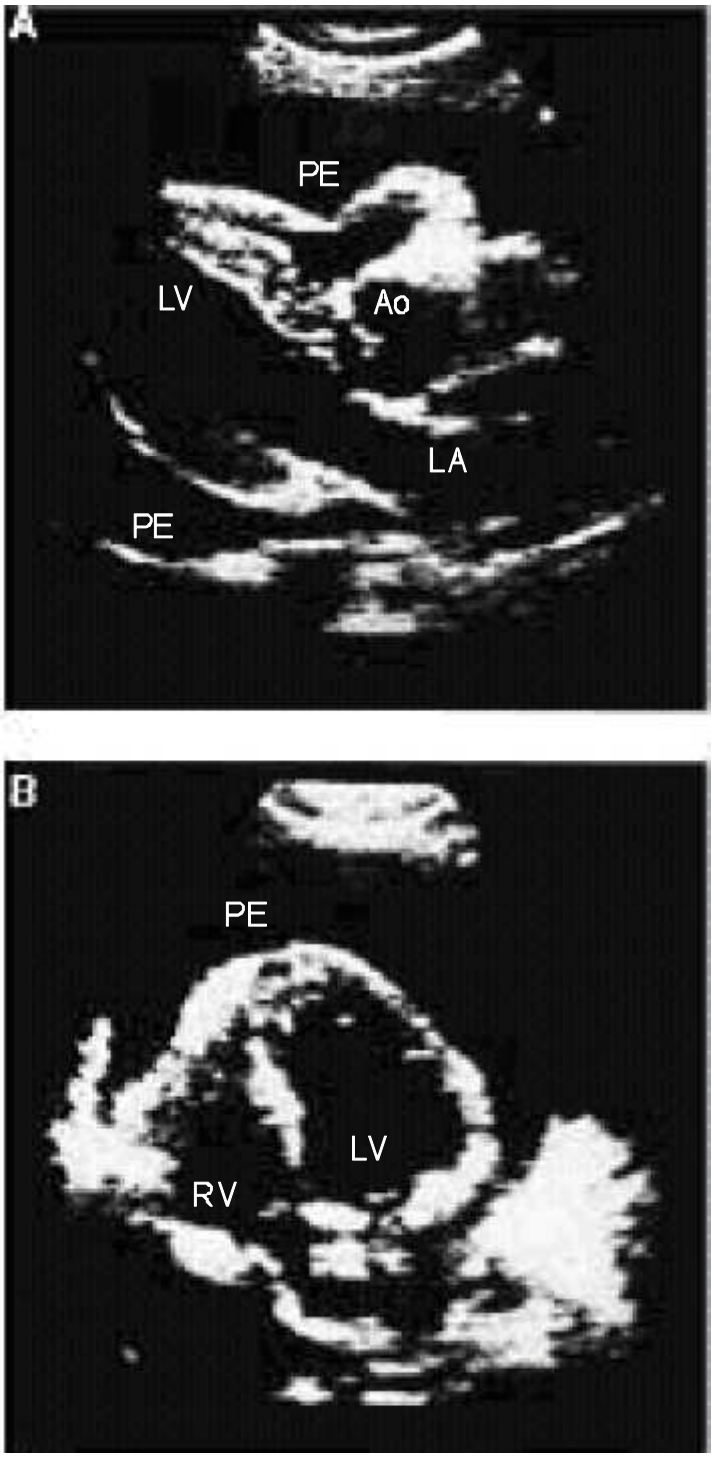


Left upper pulmonary vein pulsed wave Doppler flow pattern.

There are three waves: systolic S wave, diastolic D wave, atrial reversal A wave. This image shows an abnormal diastolic dominant pattern. Normally the S wave is larger than the D wave. In severe MR there may be another wave of systolic flow reversal.



Apical four-chamber view in a patient with a dilated cardiomyopathy and severe mitral regurgitation in ESRD



A case of Pericardial effusion - in a pt with severe CRF

K. Dis.No. 27144/E4/1/2005.

Govt. Rajaji Hospital,
Madurai – 625 020. Dt. 06.04.06.

Sub: Establishment – Govt. Rajaji Hospital, Madurai – Ethical Committee Projects approved by the Committee – Intimation – Sent – Reg.

The Ethical Committee of the Govt. Rajaji Hospital, Madurai was held at 12 Noon on 01.04.2006 at the Dean's Chamber, Govt. Rajaji Hospital, Madurai, and the following Projects were approved unanimously by the Committee Members.

S.No.	Name of the Student	Name of the Project approved
01)	Dr.G.Madhusudhanan, CRRI,Govt. Rajaji Hospital.	Diabetic Foot Syndrome.
02)	Dr. G, Ramesh, PG in MD (Gen.Med.) Madurai Medical College.	Micro albumin Urea in HIV/AIDS patients.
03)	Dr. P. Thirumalaikolundu Subramanian, Professor & HOD of Medicine.	Autonomic Neuropathy among AIDS cases
04)	-do-	Lidovudine level in AIDS cases
05)	-do-	Lactic acid levels among AIDS cases
06)	-do-	Post Traumatic stress disorder among AIDS patients.
07)	-do-	Computer knowledge and lifestyle among HCWS
08)	Dr. D. Babu Vinish, PG in MD(Gen. Med.) Madurai Medical College.	Target organ damage in hypertension.
09)	Dr. K. Sidharthan, PG in MD.(Gen. Med.) Madurai Medical College.	Serum Sodium Potassium profile in hypertensives.
10)	Dr. Revathy Janakiraman, Addl.Prof.of Obst.& Gyn. Madurai Medical College.	Changing trends in Cacsarean sections
11)	-do-	Awareness of contraceptives and HIV among unwed pregnant teenagers.
12)	Dr. V. Pavanasakumar, PG in MD(Gen. Medi.) Madurai Medical College.	Echocardiographic assessment of Cardiac dysfunction in patients of Chronic renal failure.
13)	Dr. M. Rajkumar, PG in MD (Gen.Med.) Madurai Medical College.	Optimal use of Anti-Snake venom in snake-bite envenomation.
14)	Dr.O.Chandran, PG in MD(Gen.Med.)	Socio demographic and Clinical aspects of acute diarrhoeal disease among adults.

S.No.	Name of the Student	Name of the Project approved
15)	Dr. P. Thirumalaikolundu Subramanian, Professor & HOD of Medicine, .	Injection practices among CRRIs.
16)	-do-	Specific learning disorders among HIV positive children.
17)	Dr. D. David Praveen Kumar, PG in MD(Gen.Med.)	Elderly Tuberculosis.
18)	Dr. Vipindas.C. PG in MD (Gen.Med.)	Music and Memory.
19)	Dr.M. Srinivasan, MBBS Student, Madurai Medical College.	Prevalance of Lipodystrophy among HIV/AIDS patients.
20)	Dr.E. Manivannan, PG in Pharmacology.	Cutaneous drug eruptions with special reference to non steroidal anti-inflammatory drugs.
21)	Dr. K. Baskaran, PG in Pharmacology.	Prescriptions and Doctors.
22)	Dr. S. Murugesan, PG in MD (Gen.Med.)	Congestive Cardiac failure.

Please note that the investigator should adhere the following:-

01) She/He should get a detailed informed consent from the patients/participants and maintain confidentially.
02) She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the Institution or Government.
03) She/He should inform the Institution Ethical Committee in case of any change of study procedure site and investigation or guide.
04) She/He should not deviate for the area of the work for which applied for Ethical clearance.
05) She/He should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
06) She/He should abide to the rules and regulations of the Institution.
07) She/He should complete the work within the specific period and apply for, if any extension of time is required, She/He should apply for permission again and do the work.
08) She/He should submit the summary of the work to the Ethical Committee on completion of the work.
09) She/He should not claim any funds from the Institution while doing the work or on completion.
10) She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

Dean/Chairman,
Ethical Committee, Govt. Rajaji Hospital, Madurai.

MASTER CHART

S.No.	Age	Sex	Group	Wt..	HT	Hb.	Urea	Creatinine	Cr. Cl	EF (%)	FS(%)	E	A	E/A	LVH	PE	Others
1	30	M	I	58	+	10	60	1.6	55.4	57.4	33.4	0.57	0.5	1.14	173		
2	32	M	I	60	+	11	68	2.1	42.9	61.7	36.1	0.61	0.54	1.13	178		
3	32	M	I	62	+	9.8	62	1.5	62.1	60.8	35.4	0.57	0.54	1.06	167		
4	35	M	I	65	+	9.9	80	2.1	45.1	56.5	29.6	0.73	0.51	1.43	168		
5	35	M	I	64	+	8.8	90	2.3	40.6	59.8	30.3	0.81	0.62	1.31	174		
6	36	F	I	56	+	8.5	96	2.8	30.6	62.7	35.9	0.69	0.53	1.3	140		
7	36	M	I	64	+	8.7	66	1.5	61.6	61.6	34.6	0.59	0.54	1.09	171		
8	37	M	I	68	+	9	70	2.9	33.5	58.4	30.4	0.72	0.69	1.04	169		
9	37	M	I	69	+	8.8	76	3	32.9	59.9	30.8	0.73	0.79	0.92	237		
10	38	M	I	70	+	10.1	64	2.7	36.7	62.8	35.2	0.81	0.79	1.03	230		
11	38	F	I	68	+	8.4	67	2.6	31.5	60.3	34.9	0.79	0.78	1.01	120		
12	40	M	I	69	+	8.2	80	3	31.9	47.2	24.9	0.56	0.55	1.02	232		
13	41	M	I	69	+	9.1	72	2.8	33.9	56.8	29.7	0.61	0.66	0.92	170		
14	41	F	I	68	+	8.6	60	2.2	36.1	62.1	34.8	0.74	0.53	1.4	134		
15	42	M	I	67	+	8.5	96	2.3	39.6	60.7	33.4	0.75	0.63	1.19	178		
16	42	M	I	58	+	8.8	64	2.4	32.9	57.5	32.8	0.83	0.9	0.92	180		
17	45	M	I	55	+	9.2	58	2	36.3	57.8	31.7	0.6	0.55	1.09	165		
18	48	F	I	59	+	8.1	62	2.2	30.2	58.2	32.3	0.54	0.53	1.02	124		
19	50	M	I	68	+	8.2	70	2.8	30.4	59.1	33.1	0.79	0.85	0.93	173		
20	55	M	I	62	+	8	64	2.1	34.9	60.2	33.6	0.59	0.54	1.09	238		
21	31	M	I	50	-	9.4	62	2.1	36.1	58.4	30.4	0.73	0.51	1.43	164		
22	32	F	I	54	-	8.4	68	2.3	30.9	58.3	29.9	0.81	0.79	1.03	118		
23	32	F	I	51	-	8.2	67	2.1	30.9	63.8	35.8	0.56	0.55	1.02	139		
24	34	M	I	52.5	-	9	69	2.4	32.2	62.7	35.1	0.72	0.69	1.04	175		
25	34	M	I	56	-	7.9	68	2.8	30.4	61.8	34.9	0.82	0.87	0.94	171		
26	40	M	I	59	-	7.8	70	2.9	30.3	60.9	33	0.79	0.85	0.93	169		

S.No.	Age	Sex	Group	Wt..	HT	Hb.	Urea	Creatinine	Cr. Cl	EF (%)	FS(%)	E	A	E/A	LVH	PE	Others
27	46	F	I	60	-	8.9	68	2.3	31.9	59.8	31.5	0.59	0.58	1.02	134		
28	46	F	I	59	-	8.4	60	2.1	31.2	59.9	30.4	0.61	0.51	1.2	139		
29	48	F	I	56	-	8.7	54	1.8	33.8	58.3	29.6	0.67	0.52	1.29	128		
30	51	M	I	55	-	8.2	62	2.2	30.9	60.7	32.7	0.77	0.51	1.51	178		
31	28	M	II	55	+	6.2	148	5	17.1	47.3	23.7	0.56	0.6	0.93	270		
32	29	M	II	59	+	7.8	132	4.9	17.9	60.9	33.4	0.74	0.65	1.14	260		
33	29	F	II	55	+	6.3	124	4.2	17.2	55.7	22.2	0.76	0.81	0.94	160	+	
34	30	M	II	60	+	7.9	120	4.6	19.9	54.4	29.1	0.73	0.66	1.11	190		
35	30	M	II	58.5	+	7.8	126	4.7	18.3	57.1	28.1	0.69	0.61	1.13	187		
36	31	M	II	57.5	+	6.4	140	5.1	17.1	58.8	22.8	0.48	0.51	0.94	274		
37	31	M	II	56.5	+	6.2	138	4.9	17.5	59	34.2	0.86	0.91	0.95	182		
38	32	M	II	59	+	6.1	130	5.2	17.1	42.8	22.4	0.82	0.73	1.12	244		MR
39	32	M	II	56.5	+	6	140	5.1	16.6	44.9	23.6	0.5	0.53	0.94	256	+	MR
40	33	M	II	60.5	+	6.4	142	5.2	17.3	60.7	22.7	0.74	0.78	0.95	187		
41	34	F	II	56.5	+	6.7	100	4.1	17.2	59.8	23.1	0.74	0.67	1.1	227		
42	35	F	II	57.5	+	6.2	124	4.1	17.4	60.1	23.5	0.83	0.79	1.05	235		
43	38	F	II	58	+	7.8	98	3.2	21.8	61.1	33.7	0.6	0.57	1.05	154		
44	40	M	II	64	+	8.1	140	4.2	21.2	60.7	31.2	0.73	0.61	1.2	183		
45	42	F	II	63	+	6.8	140	4.3	16.9	60.2	22.1	0.81	0.87	0.93	160		
46	43	M	II	67	+	8.2	130	4.7	19.2	59.9	30.7	0.85	0.56	1.52	274	+	
47	43	F	II	68	+	7	150	5.1	15.3	47.5	23.1	0.86	0.92	0.93	260		
48	49	F	II	69.5	+	6.6	140	5.6	13.3	45.4	22.9	0.8	0.75	1.07	253	+	
49	52	M	II	70.5	+	7	136	5.5	15.7	55.1	29.7	0.84	0.9	0.93	275		MR
50	58	M	II	68	+	6.8	147	4.9	15.8	54.9	28.4	0.85	0.6	1.42	181		
51	30	M	II	62	-	8.8	120	4.2	22.6	60.7	29	0.7	0.64	1.09	184		
52	31	F	II	63	-	8.1	128	4.1	19.8	58.8	30.4	0.63	0.61	1.03	145		
53	32	M	II	58	-	8.2	132	4.2	20.2	60.1	31.1	0.8	0.57	1.4	181		
54	38	M	II	57	-	7.6	140	4.5	17.9	59.4	22.7	0.79	0.6	1.32	190	+	
55	40	F	II	68	-	6.4	148	5.1	15.7	44.7	22	0.68	0.56	1.21	259		

S.No.	Age	Sex	Group	Wt..	HT	Hb.	Urea	Creatinine	Cr. Cl	EF (%)	FS(%)	E	A	E/A	LVH	PE	Others
56	46	F	II	62	-	6.8	128	4.2	16.4	58.3	32.1	0.74	0.79	0.94	157		
57	51	M	II	66	-	7.2	139	5.4	15.1	58.9	33.3	0.69	0.72	0.96	189		
58	52	M	II	64	-	7.8	140	4.8	16.2	60.8	30.4	0.7	0.59	1.19	187		
59	54	M	II	68	-	8.2	138	4.4	18.4	61.1	29.4	0.84	0.56	1.5	181		
60	55	M	II	71	-	8.1	128	4.3	19.5	59.9	30.6	0.63	0.61	1.03	184		
61	30	M	III	62	+	6.8	154	6.6	14.3	36.7	18.4	1	0.8	1.25	300	+	MR
62	30	M	III	58	+	6.4	148	6.2	14.2	58.5	20.5	1.01	0.6	1.68	310		
63	32	F	III	54	+	7.1	142	5.8	11.8	57.3	30	0.59	0.62	0.95	250		
64	36	F	III	41	+	6	148	6	8.4	52.2	19.5	0.97	0.61	1.59	164		
65	37	F	III	47	+	5.8	158	6	9.5	54.4	20.3	0.99	0.62	1.6	160		
66	37	M	III	51	+	6	160	6.8	10.7	56.8	29.4	0.85	0.9	0.94	200	+	
67	38	M	III	48	+	5.8	172	7.1	9.5	57.1	20.6	0.94	1.02	0.92	310	+	MR
68	39	M	III	64	+	4.8	178	8.2	10.9	58.4	29.4	0.89	0.6	1.48	320		
69	39	F	III	62	+	4.9	182	8.1	9.1	36.7	18.5	0.73	0.61	1.2	250	+	MR
70	40	M	III	53	+	5.9	142	6.2	11.8	57.2	21	0.72	0.78	0.92	190	+	
71	41	F	III	55.5	+	5.8	164	7.2	9.1	39.4	20.9	0.81	0.86	0.94	256	+	
72	42	M	III	58	+	5.9	168	7.4	10.6	37.2	20.1	0.65	0.59	1.1	194		
73	42	F	III	59	+	6.2	144	6.1	11.1	53.4	27.8	0.84	0.9	0.93	156		
74	43	M	III	64	+	6.1	142	6.2	13.9	55.7	29.3	0.79	0.64	1.23	198		
75	45	M	III	67	+	6.4	140	6.4	13.8	57.4	28.1	0.83	0.6	1.38	324		MR
76	45	M	III	54	+	5.8	176	8.1	8.8	36.9	19	0.76	0.81	0.94	300		
77	47	F	III	51	+	6.1	145	6.2	9.1	57.3	26.4	0.86	0.91	0.95	150		
78	48	M	III	56	+	5.5	180	8.7	9.1	56.7	27.1	0.74	0.79	0.94	191		MR
79	50	F	III	48	+	5.7	184	8.2	6.2	41.3	19.3	0.85	0.91	0.93	264	+	
80	52	M	III	50	+	5.2	190	8.8	6.9	40.7	18.9	0.89	0.95	0.94	316		
81	29	M	III	57	-	6.1	154	6.9	12.7	58.5	28.4	1.01	0.99	1.02	197		
82	32	F	III	54	-	6.4	160	6.1	11.2	57.9	28.6	0.97	0.59	1.64	147		
83	33	M	III	64	-	6.8	170	7.1	13.3	58.3	28.1	0.71	0.76	0.93	195		
84	33	M	III	57.5	-	7	156	6.9	12.3	57.8	27.9	0.98	0.83	1.18	189		

S.No.	Age	Sex	Group	Wt..	HT	Hb.	Urea	Creatinine	Cr. Cl	EF (%)	FS(%)	E	A	E/A	LVH	PE	Others
85	34	F	III	52	-	6.4	172	6.2	10.4	41.9	20.6	0.81	0.86	0.94	160	+	
86	36	F	III	47	-	6.9	198	7.3	7.9	57.5	20.4	0.79	0.83	0.95	324		
87	37	M	III	51	-	5.7	192	8.1	9.2	40.9	19.9	0.64	0.68	0.94	319		
88	42	M	III	62	-	6.4	200	6.1	13.8	56.9	29.1	0.72	0.77	0.94	191		
89	44	M	III	66	-	5.3	194	8.8	10.3	42.1	20.1	0.94	1.01	0.93	305		
90	50	M	III	68	-	7	156	6.1	13.9	57.4	29.8	0.97	0.86	1.13	194		
91	30	M	IV	59	-	12	18	0.4	100.2	60.6	34.9	0.64	0.49	1.31	169		
92	31	M	IV	56	-	13	19	0.6	121.1	59.4	30.4	0.62	0.41	1.51	210		
93	31	F	IV	58	-	11.8	18	0.4	124.4	58.9	29.9	0.68	0.44	1.55	110		
94	32	M	IV	60	-	11.6	20	0.6	128.6	63.4	35.3	0.67	0.42	1.6	174		
95	32	M	IV	60	-	12	18	0.4	112.5	67.9	37	0.73	0.43	1.7	185		
96	33	F	IV	58	-	11.8	19	0.8	91.6	68.9	37.1	0.69	0.44	1.57	184		
97	33	M	IV	62	-	13	18	0.6	131.6	62.4	32.2	0.64	0.5	1.28	217		
98	34	M	IV	65	-	12	16	0.4	136.7	68.1	36.9	0.64	0.47	1.36	211		
99	35	F	IV	62	-	11	19	0.6	128.1	61.5	31.4	0.74	0.48	1.54	113		
100	36	M	IV	55	-	12	20	0.6	132.4	59.5	30.9	0.7	0.51	1.37	164		
101	36	M	IV	56	-	13	18	0.6	101.1	64.3	33.6	0.69	0.45	1.53	189		
102	40	M	IV	60	-	14	26	1	92.6	66.7	34.8	0.67	0.45	1.49	207		
103	41	M	IV	64	-	12	22	1.1	125.7	68.1	36.5	0.6	0.49	1.22	196		
104	42	F	IV	72	-	11	18	0.9	108.9	60.3	30.4	0.62	0.42	1.48	179		
105	43	M	IV	59	-	11.2	24	0.8	99.4	62.1	31.7	0.71	0.48	1.48	190		
106	45	M	IV	64	-	12	22	0.8	105.6	68.2	37.1	0.71	0.45	1.58	184		
107	50	M	IV	62	-	11.8	18	0.6	129.2	59.1	29.9	0.73	0.46	1.59	167		
108	51	F	IV	52	-	10.9	21	0.7	91.1	59	30.1	0.72	0.5	1.44	154		
109	51	M	IV	68	-	12.8	26	1	105.1	60.1	31.3	0.67	0.44	1.52	176		
110	54	F	IV	51	-	9.8	18	0.4	129.4	61.9	31.7	0.61	0.43	1.42	181		
111	35	M	IV	55	-	12.8	22	0.6	133.7	64.5	34.8	0.73	0.49	1.49	169		
112	38	M	IV	60	-	12.6	18	1	106.3	66.1	35.9	0.68	0.5	1.36	174		
113	39	F	IV	52	-	11	26	0.8	103.3	68	37	0.69	0.47	1.47	149		

S.No.	Age	Sex	Group	Wt..	HT	Hb.	Urea	Creatinine	Cr. Cl	EF (%)	FS(%)	E	A	E/A	LVH	PE	Others
114	40	M	IV	64	-	12.8	24	0.6	126.9	58.9	30.4	0.7	0.45	1.56	179		
115	42	M	IV	62	-	13	22	0.4	120.6	59	30.6	0.67	0.47	1.43	174		
116	43	F	IV	48	-	10.8	18	0.8	91.6	60.4	31.5	0.62	0.49	1.27	186		
117	45	M	IV	62	-	11	21	1.1	102.3	62.2	33.6	0.74	0.48	1.54	205		
118	48	M	IV	59	-	12	16	0.8	94.2	64.7	33.4	0.73	0.42	1.74	199		
119	49	F	IV	50	-	11	18	0.6	107.4	68.4	36.9	0.65	0.41	1.59	144		
120	50	M	IV	62	-	12	21	1	96.9	60.9	32.2	0.69	0.45	1.53	153		

Group I Patients with creatinine clearance of 30 – 90ml/min/1.73m^2

Group II Patients with creatinine clearance of 15 – 29 ml/min/1.73m^2

Croup III Patients with creatinine clearance of < 15 ml/min/1.73m^2

Group IV Controls

HT Hypertension

Cr. Cl. Creatinine clearance

EF Ejection Fraction

FS Fractional Shortening

E Early diastole velocity

A Peak atrial filling velocity

LVH Left Ventricular Hypertrophy

PE Pericardial effusion

MR Mitral regurgitation